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ORAL SURGERY
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OPERATIVE ORAL SURGERY

MULTIPLE FACIAL FRACTURES

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SEVERE injuries to the facial skeleton as a result of both industrial and automobile accidents are frequently seen today. Whatever the cause, injury to the facial area may produce skull fractures as well as other injuries. The clinical picture can become so complicated that its management requires the services of the neurologist, the general surgeon, the orthopedic surgeon, the internist and the oral surgeon. In one of the cases to be reported in this article, the patient fell 60 feet from the door of a hanger at Idlewild Airport. The face and head injuries were the most severe, but there were also some injuries to the right knee and tarsal bones. In the second case the patient was struck by an automobile and suffered cranial and facial injuries as well as a fractured arm. It was remarkable that either patient survived the initial impact and was able to undergo the involved surgical procedures necessary to restore function and appearance.

CASE 1.—A 27-year-old white man was seen at St. Joseph's Hospital in Far Rockaway, New York, on May 13, 1958, following a fall from the door of a hangar at Idlewild Airport.

Examination.—The entire facial area was markedly swollen, and the bridge of the nose was oblitered beneath the edema of the eyes and cheeks. The right eye exhibited scleral ecchymosis, and a mixture of spinal fluid and blood dripped from the mouth, nose, and left ear. The patient was in acute distress, with difficult breathing and severe pain, and was bordering on shock. There was a history of a loss of consciousness, but both pupils reacted equally to light. The patient responded logically and was well oriented as to time and place both before and after the accident. The pulse rate was 100, and blood pressure was 95/70. Despite the absence of signs of increasing intracranial pressure, it was apparent that the patient was in acute distress and rapidly deteriorating. It was a tribute to the physician** who had rendered initial care that the patient was even alive at this point.

After a conference with this physician, it was agreed that an immediate tracheotomy was necessary if we were to control oral hemorrhage and prevent respiratory difficulty. It

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was obvious that replacement of lost blood would not stabilize the patient's condition unless hemorrhage was controlled.

Emergency Procedure.—The patient was moved to the operating suite where, under local anesthesia, a tracheotomy was performed and the airway vastly improved. Debris, blood, and spinal fluid were suctioned from the oral cavity, and the shattered maxilla was visualized. The fractured alveolar process, with fragments of attached teeth, had been driven upward into the antrum. Careful removal of shattered bone, teeth, and other debris disclosed a fracture of the cribriform plate of the ethmoid bone through which cerebrospinal fluid seeped. The medial and lateral walls of both antra were shattered, and the entire maxilla actually hung from shreds of soft tissue (Fig. 4).

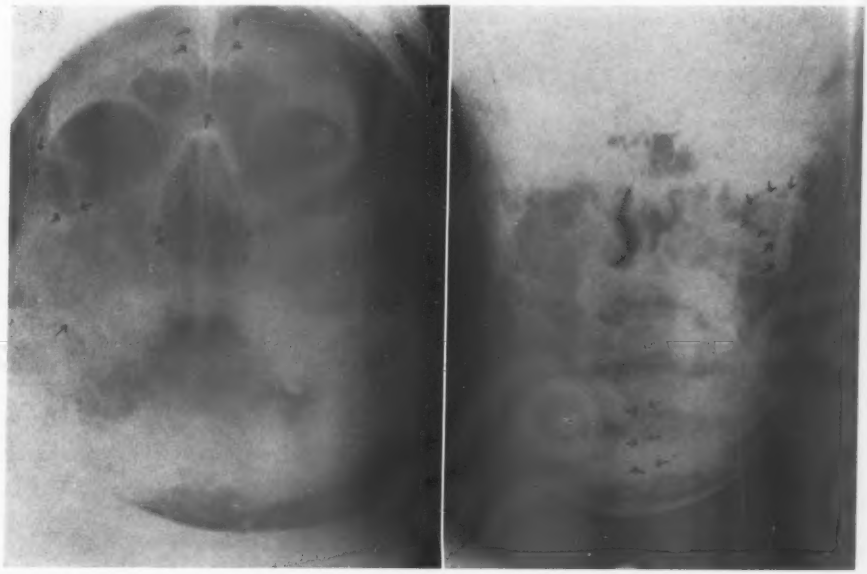

Fig. 1. Fig. 2.

Fig. 1.—Waters' position roentgenogram taken on May 20, 1958, showing severe comminution in middle facial area.

Fig. 2.—Posteroanterior view of mandible showing severe fractures at symphysis, right angle, and left temporomandibular joint.

The upward and backward impaction of the maxilla had driven the perpendicular plate of the vomer into the cribriform plate of the ethmoid bone. By disimpacting the maxilla, it was possible to pull the vomer out of this region, thus decreasing the opening and eventually permitting fibrosis to seal off the leak.

It was also noted that the palatal vault was fractured through its midline. The maxilla was molded into some approximation of its original shape after the soft-tissue lacerations were closed. The antra were not packed since it was felt that the pooling of infected blood and spinal fluid would constitute a far greater hazard of possible meningitis. This would provide an incubation area from which bacteria could ascend into the anterior cranial fossa. The danger would be somewhat less if the fluids were allowed to drain through the sinus, away from the cranial injury, until fibrosis sealed off the cribriform plate fracture. The closure of all soft-tissue lacerations materially lessened the bleeding and rhinorrhea.

Another fracture in the symphysis was exposed when the loose lower anterior teeth, with their attached alveolus, were removed (Fig. 3). The mucous membrane was closed

over this area and sutured. Multiple lacerations in the lips, cheeks, tongue, and floor of the mouth were repaired after portions of tooth debris were removed. A compound fracture was noted in the right third molar area as the soft tissues in this region were closed (Fig. 3).

Control of bleeding was, of necessity, the first step in stabilizing the patient's condition. It was now possible to replace the lost blood and electrolytes in a more exacting manner and thus reverse the shock which was slowly developing.

Postoperative period: The next few days were stormy ones. The patient became anuric and then incontinent. Finally, an indwelling catheter was placed and the urinary problem was stabilized. The temperature rose to 104° F., despite administration of Combiotic, Achromycin, glucose, blood, and other supportive measures. It was not possible to control pain with morphine derivatives, since these drugs would increase the intracranial pressure. To add to an already complicated picture, an allergic reaction to penicillin occurred. The antibiotics were changed a number of times during the first week until cosatetragen was found suitable. Finally, on the eighth hospital day, the entire picture stabilized. The cerebral spinal fluid ceased flowing from the nose and the ear. In view of the patient's improved condition, it was felt that reconstruction of the mandible could be undertaken in about one week.

It was now possible to have a complete x-ray examination made. The following fractures were found as the result of this survey:

Mandible (Figs. 1 and 2)

1. Fracture of symphysis
2. Fracture of alveolar plate of symphysis
3. Fracture of right angle with severe comminution and impaction
4. Fracture of left mandibular condyle with medial displacement

Maxilla (Fig. 1)

1. Fracture of midpalate
2. Fracture of right palatine bone
3. Fracture of left palatine bone
4. Fracture of right palatine process of maxilla
5. Fracture of left palatine process of maxilla
6. Fracture of left medial pterygoid plate of sphenoid
7. Fracture of left lateral pterygoid plate of sphenoid
8. Fracture of right medial pterygoid plate
9. Fracture of right lateral pterygoid plate
10. Fracture of horizontal portion of left palatine bone
11. Fracture of horizontal portion of right palatine bone
12. Fracture of right lateral maxillary wall
13. Fracture of left lateral maxillary wall
14. Fracture of right medial maxillary wall
15. Fracture of left medial maxillary wall
16. Depressed fracture of left orbital floor
17. Separation of maxilla from skull

Malar (Fig. 1)

1. Fracture of right frontomalar suture
2. Fracture of right orbital floor through infraorbital canal
3. Fracture of right zygomatic arch

Vomer (Fig. 1)

1. Fracture of vomer upward into ethmoid bones

Ethmoid (Fig. 1)

1. Fracture of cribriform plate

In view of the severity and number of fractures, it was decided that several surgical procedures should be performed.

Second Operation.—Reconstruction of the mandible was the first oral surgical procedure indicated. It was decided to begin this by assembling the fractured parts with a Roger Anderson appliance. On May 28, 1958, while the orthopedic team removed the fractured

Fig. 3.

Fig. 4.

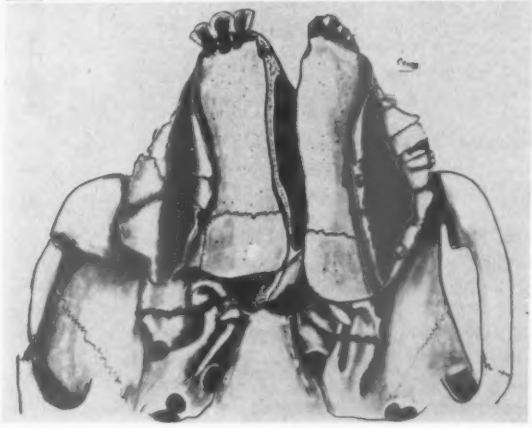

Fig. 3.—Drawing of skull showing relationship of fractures to x-ray diagnosis (Figs. 1 and 2).

Fig. 4.—View of maxilla showing extent of injury.

patella, two pins were placed on either side of the symphysis fracture. Next an attempt was made to place several pins in the right ramus, but these could not be maintained because of insufficient bony thickness.

Several days later, when the discomfort from the orthopedic procedure had lessened, 5 pounds of traction was applied to the facial appliance. It was hoped that traction would disimpact the shattered right angle and pull the left condylar neck down from the temporal rim of the condylar fossa. Both of these ends were achieved to a degree. The results were not completely satisfactory, however, and it now became necessary to reduce these elements by means of an open operation (Fig. 5).

On June 12, 1958, a plaster headcap was constructed. Sufficient stabilization bars were included, so that fixation of the mandible could be made in any of several directions (Fig. 7).

Fig. 5.

Fig. 6.

Fig. 5.—Posteroanterior film of mandible taken on May 30, 1958, showing Roger Anderson appliance in position in symphysis.

Fig. 6.—Posteroanterior film taken on June 14, 1958, showing mandible after open reduction of right angle. Note improved position of left condylar neck.

Third Operation.—On June 14, 1958, the right side of the face was prepared in the usual manner and the area was suitably draped. An incision approximately 4 cm. in length was made below the lower border of the mandible. The dissection was carried through the skin, fascia, and platysma. The anterior facial artery and vein were located, exposed, and ligated. An incision was made through the periosteum at the lower mandibular border, and the masseter muscle, parotid gland, and seventh nerve were reflected. The severely comminuted fracture was thus exposed. One fragment was removed and the remainder was assembled with a wire suture to maintain proximation. The wound was closed in layers and the mandible was reattached to the headcap in the new position (Fig. 6). The postoperative roentgenogram showed the buccal and lingual fragments to be impacted and overlapping. This was not an ideal arrangement, but it would permit union by direct bone contact. The left condylar neck had fused to the rim of the temporal portion of the articular fossa. However, in estimating the loss of height in the right ramus, we decided that the left condyle could be reshaped to fit the fossa and thus equalize the loss on the opposite side. It was hoped that this would result in a symmetrical mandible that would function adequately.

One month later the mandible was tested for function, and the left temporomandibular joint was found to be ankylosed. It now became necessary to open the temporomandibular joint and refit the stump of the condylar neck to the fossa.

Fourth Operation.—On July 2, 1958, the field was suitably prepared and draped. A preauricular incision was made in the crease of the left ear from the tragus to the level of the helix, where it was angulated anteriorly approximately 30 degrees for an additional 2 cm. The skin dissection was deepened through the superficial fascia to the parotid fascia.

Fig. 7.—June 12, 1958. Weights applied to headcap via connection to Roger Anderson appliance.

The superficial temporal artery and vein were exposed and ligated. Next the zygomatic branch of the facial nerve was located and preserved. The temporomandibular capsule was opened, and the fusion of the neck of the condyle to the zygoma was separated. A portion of the neck was then recontoured and shaped to fit the mandibular fossa. The mandible was then rotated to the opposite side to permit inspection of the medial aspect of the condylar neck. Because of the age of the injury and the sealing-off of the blood supply from the internal maxillary artery, no bleeding was encountered. The fractured condylar head was found fused to the medial aspect of the ramus below the coronoid process. This proved difficult to separate, but finally the condylar head was removed. The condylar fossa was closed in layers, and the remainder of the wound was similarly closed. The mandible was again fixed to the headcap, and after three days movement was permitted. This was encouraged by adding traction to the symphysis and having the patient exercise against progressively increasing weights (Fig. 7). This program helped so much that the Roger Anderson pins were left in situ until July 25, 1958.

An intraoral stent was constructed to fit the mandible. This was provided with a place from which weights could be suspended, so that the exercise program could be continued after the pins were removed.

Fifth Operation.—On July 25, 1958, we decided to remove the Roger Anderson pins which had been placed on May 28, 1958. It was felt that at the same time the maxillary and mandibular alveolar process could be examined, all fractured roots could be removed, and the mouth could be prepared for dentures. The patient was draped in the usual manner after suitable preparation of the face and mouth. An incision was made along the entire maxillary ridge, from tuberosity to tuberosity, and the mucous membrane was elevated. A number of fractured roots and fragments were removed, and the ridge was properly contoured for a denture base. The tissues were approximated with 000 silk.

Fig. 8.

Fig. 9.

Fig. 8.—Waters' position roentgenogram taken in January, 1959, showing healed middle facial area.

Fig. 9.—Posteroanterior film of mandible in January, 1959, showing bone continuity and union.

The entire mandibular tooth-bearing area was exposed in a similar way through an incision from the right third molar area to the left. A number of fractured teeth and fragments were removed, and the ridge was contoured for a denture base. The tissues were closed with 000 silk.

Next, the Roger Anderson pins were removed with some difficulty in the region of the symphysis but with no difficulty in other areas. After this procedure the face was properly cleansed and protected with dressings.

There was no great reaction to this extensive procedure during the next two or three days. The sutures were removed on the fifth postoperative day, and the patient was continued on Cosatetragen for two more days.

The patient was finally discharged from the orthopedist's care about Sept. 15, 1958, after four months in the hospital. Healing progressed uneventfully, and the patient was referred to his dentist for the construction of dentures. These were completed by early October, 1958, and satisfactory function was obtained.

The patient remained under observation for the next eight months, and mandibular function was found to be almost normal. A slight deviation of the mandible to the

condylectomy side was corrected by proper exercise, and the bulge of the right mandibular angle was slowly resorbed. Some discomfort was noted in this area, and plans were made to remove the wire and reduce facial contour if this should prove necessary.

Fig. 10. Fig. 11.

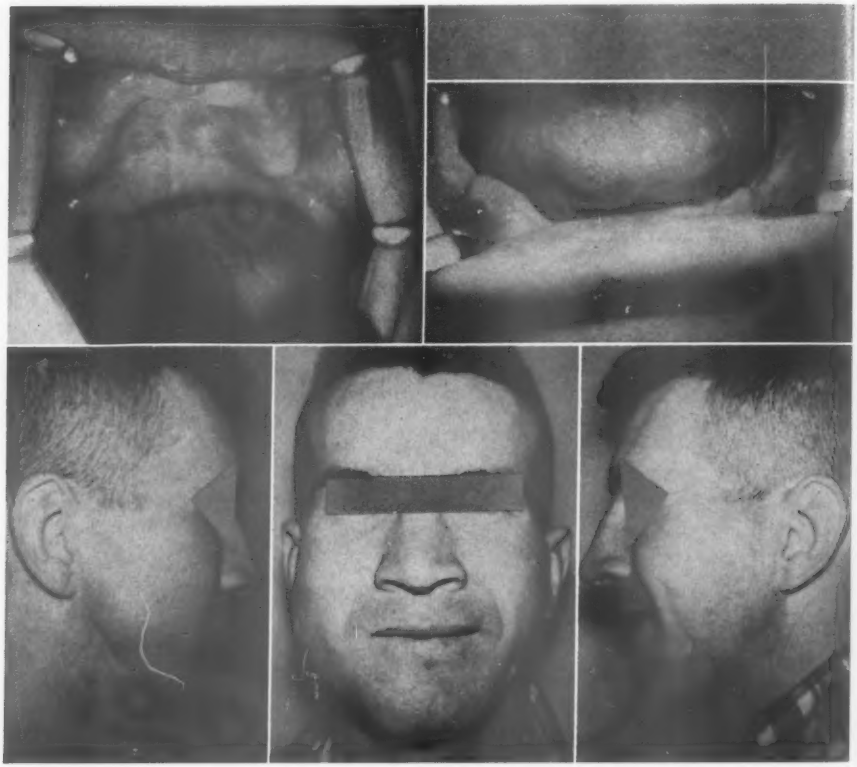

Fig. 12.

Fig. 10.—Intraoral photograph of healed maxilla in February, 1959. Note preservation of ridge contour.

Fig. 11.—Intraoral view of healed mandible in February, 1959.

Fig. 12.—Facial views of patient in March, 1959, prior to correction of prominence of right angle.

CASE 2.—A 52-year-old white man was admitted to Morrisania City Hospital on March 5, 1959, for treatment of injuries sustained in an automobile accident. Both sides of the face were tremendously swollen, and a number of lacerations were noted. There was marked facial deformity, with mandibular deviation to the left and a mucoserous discharge from both nostrils. Marked periorbital edema with subconjunctival hemorrhage and a prolapse of the bulbar conjunctiva through the palpebral fissure were suspected since the level of the left eye was considerably below that of the right. The buccal mucosa of the left cheek was ecchymotic, and all remaining teeth were loose. The intraoral opening was restricted to approximately 5 mm., and crepitus was noted on movement.

The patient was conscious and oriented but complained of diplopia and numbness in the left eye. He appeared to appreciate all tactile sensation in the upper and lower extremities.

The neurologist felt that the picture was one of cerebral concussion but that the patient should be followed closely for any signs of increasing cerebral compression.

X-ray Examination (Figs. 13, 14, 15).—Examination of the skull revealed an irregular linear fracture, approximately 11 cm. in length, extending from the frontal region posteriorly into the parietal region on the left side of the calvaria. Free air was present in the skull.

There was a fracture through the right subcondylar region of the left body of the mandible, 3 cm. from the symphysis, and another was seen immediately cephalad to the angle. All fragments were widely separated.

Other findings were as follows:

Maxilla

1. Fracture of midpalate
2. Fracture of right palatine bone
3. Fracture of left palatine bone
4. Fracture of right palatine process of maxilla
5. Fracture of left palatine process of maxilla
6. Fracture of left medial plate of sphenoid
7. Fracture of left lateral plate of sphenoid
8. Fracture of left medial plate of sphenoid
9. Fracture of left lateral plate of sphenoid
10. Fracture of horizontal portion of left palatine bone
11. Fracture of horizontal portion of right palatine bone
12. Fracture of right lateral maxillary wall
13. Fracture of left lateral maxillary wall
14. Fracture of right medial wall of maxilla
15. Fracture of left medial wall of maxilla
16. Depressed fracture of left orbital floor
17. Pyramidal fracture of middle facial area from skull

Malar

1. Fracture of left zygoma
2. Fracture of right zygoma
3. Depressed fracture of left malar bone
4. Depressed fracture of right malar bone

Nasal

1. Fracture of right nasal bone
2. Fracture of left nasal bone

Vomer

1. Fracture in region of ethmoids

Frontal

1. Orbital process of left frontal bone separated from malar frontal junction

Sphenoid

1. Greater wing of the sphenoid into medial cranial fossa

X-ray examination of the left wrist disclosed a fracture of the lower portion of the shaft of the radius with extension to the articular surface of the radius. In addition, there was a fracture of the lower portion of the ulna with dorsoposterior displacement of the distal fragment.

Treatment.—The patient was taken to the ward in critical condition, with a blood pressure of 90/70, a pulse rate of 100, a respiration rate of 26, and a temperature of 101° F. Severe pain made the patient unmanageable, but paraldehyde produced prompt sedation without additional excitement. Blood typing and crossmatching had already been done in the emergency room, and a transfusion had been started. Tetanus antitoxin and Combiotic were also administered. Vital signs improved during the night, despite the respiratory stertor caused by the fractured nasal apparatus. On the following day gradual improvement occurred, periods of uninterrupted sleep lengthened, and the patient was able to take

liquids. The Combiotic was continued; buccal Varidase was given in an attempt to reduce the facial swelling, but it did not alter the course of the edema to any marked degree. The fractures of the radius and ulna were reduced by the orthopedist, and several days later the first definitive oral surgical procedures were considered. The patient showed a marked sensitivity to any of the depressant alkaloid drugs; therefore, they were avoided. The only preoperative medication given was 50 mg. of Phenergan hydrochloride.

Fig. 13.—Drawing of facial bones showing extent of fractures in skull, maxilla, and mandible.

First Operation (March 11, 1959).—Anesthesia was induced, an endotracheal tube was inserted, the face was suitably prepared, and the region of the left mandible was draped. A 4 inch incision was made below the lower border of the mandible, and the dissection was deepened to the platysma muscle. This muscle was incised in the direction of the skin incision, and all bleeding vessels were clamped and tied. The anterior facial artery and vein were located anterior to the masseter muscle, ligated, and severed. The lower border of the mandible was located, and the periosteum was incised and elevated with the masseter muscle, parotid gland, and seventh nerve. The fractured parts were approximated, holes were drilled on either side, and a stainless steel wire suture was placed. The anterior fracture was located beneath the symphysis and realigned, and a stainless steel wire suture was placed to maintain the reduction. After this was accomplished, the periosteum was closed and the remainder of the wound approximated in layers. The opposite side of the face was now prepared and draped so that the right mandible was exposed. A 3 inch incision was made in the region of the right mandible. A dissection similar to that completed on the left side was carried out on the right mandible, but the region of the condylar neck was exposed, approximated, and wired (Fig. 16).

Postoperative care: The patient was returned to his bed awake and complaining of pain. The pain was controlled with 1,600 to 2,000 mg. of Equanil and aspirin in adequate

Fig. 14.

Fig. 15.

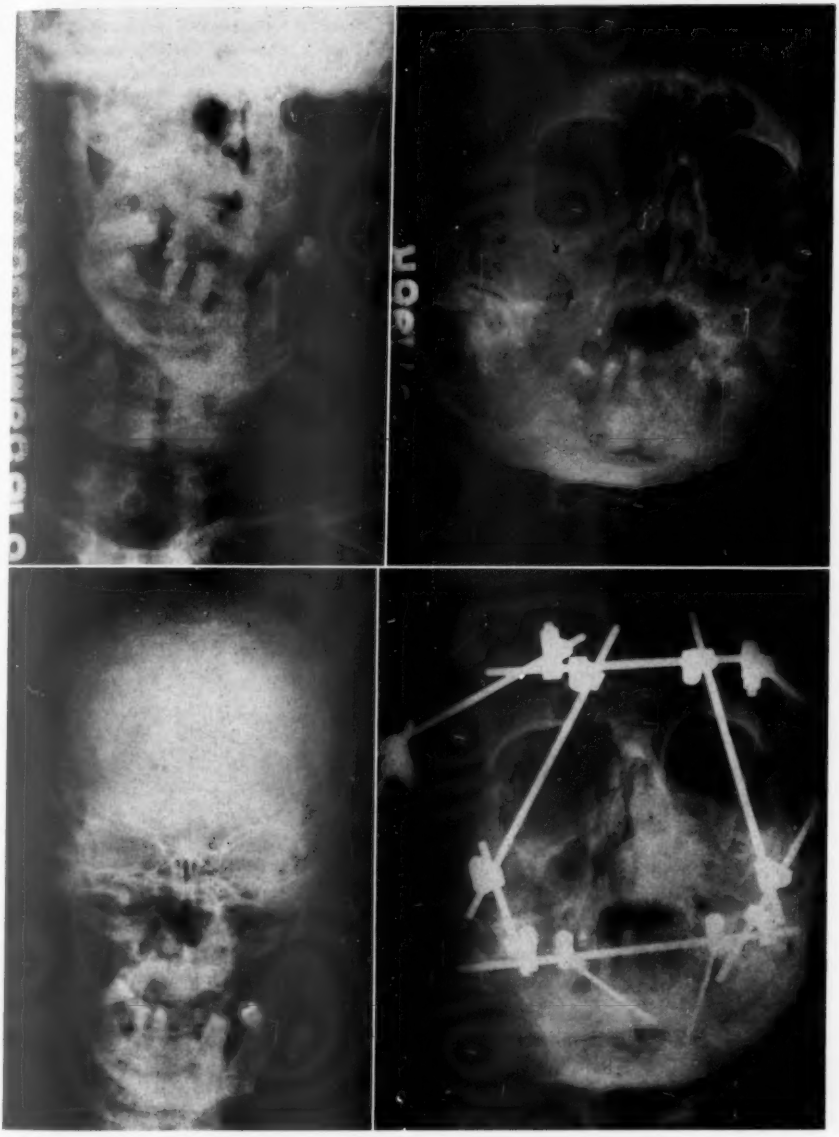

Fig. 16.

Fig. 17.

Fig. 14.—Posteroanterior roentgenogram, taken on March 5, 1959, showing severe comminuted mandibular fractures.

Fig. 15.—Waters' position film taken on March 5, 1959, showing severe middle facial fractures.

Fig. 16.—Reduction of fractures in mandible by intraosseous bone wiring.

Fig. 17.—The floor of the left orbit has been elevated and the mandible fixed with a Roger Anderson set connected to a plaster headcap.

dosage. Early mobilization of the patient out of bed and his participation in the daily routine of the hospital served to prevent chest complications. Approximately one week later, it was decided that the floor of the left orbit should be elevated in an attempt to reposition the globe of the eye. It was felt that manipulation of the left zygomatic arch might lead to distraction of the skull fracture in the temporal bone. Therefore, no attempt would be made to reposition this arch, in view of the minor or negligible deformity. It was also felt that immobilization of the mandible was necessary. For this purpose, a plaster headcap was fitted and plans were made to place in the symphysis a set of Roger Anderson pins which would be connected to the cranial anchorage.

On March 20, 1959, the patient was premedicated with 50 mg. of Phenergan hydrochloride and sent to the operating suite.

Second operation (March 20, 1959).—The patient was anesthetized, an endotracheal tube was placed, the face was prepared, the mouth was cleansed and the patient was suitably draped. Two Roger Anderson pins were embedded in the symphysis, and suitable connectors were attached for immobilization to the headcap.

The mouth was entered and the region around the endotracheal tube was packed with gauze. An incision was made along the crest of the ridge from the right cuspid region to the left tuberosity, and the flap thus outlined was elevated. The region of the severely comminuted antral wall came into view. Several of the fragments were removed, and the antrum was entered. Suction of the contents of the antrum showed the orbital floor to be markedly displaced downward and severely comminuted. The debris was removed and the antrum was packed upward with 1 inch iodoform gauze until the globe of the eye was seen in a more normal position, the orbital rim was felt to have been repositioned, and the malar prominence was once more apparent. The zygomatic arch on that side and the frontomalar junction were not manipulated because of their proximity to the skull fracture in the temporal region. Next, an opening was made through the lateral nasal wall beneath the inferior nasal concha and the antral dressing was brought out through the nose. This would permit the oral wound to close, and the antral packing could be removed through the nose. Following this, the mandible was immobilized by connecting the Roger Anderson pins to the headcap (Fig. 17).

Postoperative course: The postoperative course was uneventful. The eye remained at the proper level, and the diplopia was resolved. The infraorbital paresthesia slowly lessened, and as the intraocular hemorrhage cleared normal vision was restored. On the tenth postoperative day the packing was removed from the nose and the patient's condition was markedly improved.

The mandibular incisions did not heal uneventfully. Both incisions drained a purulent material, which culture proved to contain *Staphylococcus aureus* and *Bacillus coli.* These organisms were resistant to penicillin and streptomycin but only moderately sensitive to gantrax and Chloromycetin. The right incision closed without difficulty after several days of Chloromycetin therapy. The superior portion of the incision on the left side continued to drain for several weeks. A number of cultures identified the organism as *Staphylococcus aureus.* The *Bacillus coli* apparently was eliminated by the previous antibiotic coverage.

Periodic x-ray examination of the left mandible showed some destruction of bone around the fracture sites. It was felt that immobilization would permit union in these areas, once bacterial infection was eliminated. During the sixth week of hospitalization a series of sterile cultures were obtained from the operative site, but fixation was maintained for an additional month before function was permitted.

Third Operation.—On May 15, 1959, the decision was made to remove the wires in the left body of the mandible and to saucerize the area of infected bone. This was deemed safe, since several sterile cultures had been obtained. On operation, a definite fistula was excised, the intraosseous wires were removed, and the infected bone was saucerized. The skin of the facial wound was undermined for a considerable area and closed separately over the depressed scar.

Postoperative course: The operative site appeared to heal uneventfully until two weeks later when drainage again appeared. Cultures identified the organisms as *Staphylococcus aureus* and *Bacillus coli*. These organisms represented contaminations which had somehow invaded the surgical site despite all sterile precautions taken at the time the wound was dressed.

Fig. 18.

Fig. 19.

Fig. 18.—Lateral jaw film showing area of breakdown after removal of interosseous wires.

Fig. 19.—Lateral jaw film taken on Aug. 20, 1959, shows healing following removal of sequestra.

Sixteen weeks from the date of admission the patient was discharged, to be followed at the office. On July 17, 1959, a sequestrum was removed from the lower border of the left mandible (Figs. 18 and 19) and the area was allowed to close. The mandible was a good functioning unit, and all lateral movements were present, indicating that the coronoid processes were not limited in excursion by the untreated fracture of the zygomatic arch. Finally, the mouth was prepared for dentures by the elimination of all abnormalties in the ridges. When seen several months later, the patient was functioning well with his new dentures.

CONCLUSION

Cerebrospinal fluid leaks from the anterior cranial fossa do not always cease spontaneously under these circumstances. The neurosurgeon may decide to repair these openings with fascia-lata grafts. There is a difference of opinion as to when a middle-face injury with cribriform plate involvement may be manipulated. This becomes a matter of judgment in which the patient's general condition, neurologic status, and chances of improvement must be evaluated.

Two cases of severe facial injury have been presented. In Case 1 it was decided that the middle facial injury should be manipulated at the first treatment in order to stop cribriform plate leakage. In Case 2 the injury was not manipulated but was allowed to heal spontaneously.

The treatment of the bone injuries was completed in several stages over a number of weeks.

The problem of the hospital staphylococcus was encountered in both cases but eventually was controlled by suitable changes in drug therapy. It is suggested that the laboratory conclusions reached by sensitivity tests are not always reliable for in vivo treatment.

The use of Roger Anderson pin fixation was indicated in both cases. I do not especially like this appliance, because of the necessary postoperative care involved. In selected cases, however, it may provide an excellent method of supplemental treatment.

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BURNS OF THE ORAL CAVITY

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BURNS of the oral cavity are identical to burns elsewhere on the body, with the obvious exception of sunburns. There are slight variations, however, in their treatment. Many of the serious and complicated sequelae in the treatment of burns will not be mentioned here because of the limited scope of this article.

ETIOLOGY

The etiological factors of thermal, chemical, radiation, and electrical burns hold true for the oral cavity the same as for burns elsewhere. Classification as first-, second-, and third-degree burns also is the same as for the rest of the body.

Thermal Burns.—Thermal burns can be subdivided as to those caused by the extremes of heat and cold on either side of the body temperature. The extreme cold latent in a piece of metal placed in the mouth or on the lips can cause a surface destruction of the mucous membranes by an instantaneous adherence and freezing which causes the victim inadvertently to pull away from the object causing the discomfort. This, in turn, strips the surface tissue away, leaving a raw and sometimes bleeding surface. Usually enough epithelial structure remains, and healing takes place spontaneously. Deeper freezing causes a more serious wound which may necessitate grafting to prevent scar formation.

When heat is the malefactor, whether it is due to friction or is latent in an object, the trauma can range from a mild hyperemia to a marked destruction of tissue necessitating plastic repair at a later date.

Chemical Burns.—Chemical burns resulting from acids or alkalies should be neutralized as soon as possible with an agent that will not be destructive to the normal tissue. Continuous lavage until one is certain that no more harm will come from the destructive agent should be carried out very cautiously and thoroughly.

It should be emphasized that the swallowing of acid or alkali, causing marked destruction of the esophagus and stomach and resulting in severe shock and probable death, will not be dealt with here because of the brevity of this

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article. The slough of a burn of the mouth in this instance should be removed as soon as possible; when healthy granulation tissue is present, a split-skin graft may be applied.

Radiation Burns.—Radiation burns from x-rays or radium are best treated with watchful expectancy and supportive treatment. They should be allowed to heal before a plastic repair is attempted. The amount of destruction of surface tissue, vascular disturbance, resulting fibrosis, and deformity determines the subsequent procedures.

Electrical Burns.—Electrical burns of the oral cavity occur more frequently in infancy than at any other time of life. As soon as babies are allowed to crawl, this hazard becomes manifest. The infant's inherent tendency to place everything in the mouth and chew on it is the primary cause of such burns. When he puts an electrical cord into his mouth this chewing continues until a direct short is established between the two wires in the cord; this causes a flash burn from the arcing and also causes a surge of current to pass through the tissues, producing many changes. These changes range from a mild hyperemia to a black char, depending on the length of time the current is on, the direction of flow of the current, amperage, voltage, and the ground. Needless to say, the victims of such burns are thrown into shock and should be treated immediately.

To date, this danger has not been given enough thought and action. An electrical code should make obligatory the following specifications: (1) all lamp cords should be made of a heavy, solid rubber covering that will not break down on being chewed by an infant; (2) all wall and lamp plugs should be operated by a master switch on the wall, so that all cords and plugs are devoid of current when this switch is open; (3) all wall plugs should be about 3 feet from the floor and of a type that an infant or small child could not tamper with. These three precautions would contribute greatly toward the prevention of catastrophes.[7]

TREATMENT

In some respects, the treatment of burns of the oral cavity is a great deal more simple than that of intensive body burns. The application of voluminous dressings with pressure and other methods of therapy are not the treatments of choice here.

The most important factors in the treatment of burns of the oral cavity are (1) control of pain, (2) prevention of shock, (3) reduction of infection to a minimum, (4) active treatment of the denuded area to promote growth of granulation tissue, and (5) plastic repair of skin, muscle, and mucous membrane at the proper time.

Control of Pain.—Pain produced by a burn is continuous unless treated. The use of large amounts of morphine is a valuable factor in the control of pain. The pain factor alone will throw the patient into shock if not controlled. The dosage of any drug to control pain is directly proportional to the extent of the body burn. A Nembutal suppository of ½ to 1 grain in an infant may be adequate; one aspirin tablet may be sufficient for a child with a mild burn; or ¼ grain of morphine may suffice for an adult. This dosage will be entirely

inadequate in relieving the pain caused by a severe burn. Three or four times this dosage in the case of an extensive burn, considering the age and weight of the patient, will not be dangerous and may help substantially in preventing shock.

Prevention of Shock.—Shock may be defined as the measure of the patient's ability to withstand harmful influence from without. Fortunately, burns of the mouth are not generally accompanied by profound shock.

A burn of the oral cavity is sustained rapidly when a baby, chewing a lamp cord, creates a short circuit. The current does not pass through the entire body; the damage is local in the tissues of the mouth, and the central nervous system is spared. Therefore, it is unusual for respiratory or cardiac failure to follow. Shock, however, is a measure of damage and must be considered seriously. An infant who receives an electrical shock as a result of chewing a lamp cord usually is on a dry floor and, therefore, relatively safe; if he were in a tub of water, he would probably die instantaneously.

Shock should be treated by the following methods, when indicated: (1) control of hemorrhage, (2) restoration of body heat, (3) restoration of fluid balance by plain water, physiologic salt solution, plasma, or whole blood, (4) positioning of the patient so that his head is low and his feet and body are elevated, and (5) close watch over the patient (particularly with respect to fluid intake and output, respiration, pulse rate, and temperature) to guard against an ensuing infection.

Reducing Infection.—The currently used antibiotic drugs, such as penicillin, Aureomycin, Terramycin, and others, surely have lightened the burden of treatment and shortened the course of invalidism. These drugs reduce infections to an absolute minimum and allow granulations to form quickly.[8]

Promotion of Granulation Tissue.—To promote early stimulation of healthy granulation tissue, complete débridement of the burned area as soon as possible is advisable. Frequent irrigations with physiologic saline solution are helpful also. Where skin, muscle, and mucous membrane have been destroyed, particularly in the upper or lower lip (or both), it is recommended that these parts be allowed to heal before plastic repair is attempted. Adhesive-tape straps diligently and strategically placed with regard to future healing, muscle action, cosmetic result, relaxation of skin, and minimum scar tissue formation will help to reduce the convalescent period. Areas of destruction of the lower lip can often be corrected by the use of adhesive straps alone. These tissues are extremely friable and will not lend themselves to immediate suturing; an adhesive strap will save tissue, give good approximation, and produce a fair end result.

Plastic Repair.—Burns which destroy the vestibule of the upper or lower jaw and cheek sometimes can be corrected immediately by placing a split-skin graft over a stent of modeling compound or acrylic and joining it in place. This, however, can be done only in older and more cooperative patients. In infants the wound must be allowed to heal, with correction made subsequently.

The typical electrical burn in infants destroys the corner of the mouth, involving the skin, muscle, and mucous membrane. This type of burn should be

allowed to heal. Very often 50 per cent or more of the orifice of the oral cavity will be destroyed. When this wound has healed, it will be found that much of the body of the lip in that region has become fibrotic as a result of the surge of current through the tissues. Every effort should be made to conserve this tissue and thus reduce the deformity. Frequently, it can be utilized in the reconstruction and, with proper preparation of flaps and suturing, a good cosmetic result can be obtained. Often we find that one-half or more of the upper and lower lips has been destroyed and that, in healing, the oral cavity has become completely occluded except for, possibly, a small opening about the size of a lead pencil. These deformities should be corrected by making horizontal incisions in the cheeks, with the openings on each side balanced. Elevation of skin and mucous membrane surfaces and approximation of them will effect a good correction. The skin and mucous membrane at the corners of the newly formed mouth should be well undermined, so that the corner of the mouth is thin and has a normal appearance. If this is not done, the corners of the mouth are too heavy, they do not look normal, and they result in more contraction and narrowing of the orifice. When a narrow band of dense, fibrous tissue develops in the process of healing, it is sometimes advisable to remove it, provided that its removal does not produce a deformity. Simple incision will result in less scar tissue formation and less deformity.

Discrepancies in the lip tissues, where tissue is lost or where there is too much tissue, causing an irregularity in the vermilion border or in the mucocutaneous junction, can be corrected by the typical V-Y plasty. Several of these transpositions will be necessary to correct badly burned mouths. Destruction of large masses of tissue can be corrected only by means of the pedicle tube. In spite of its disadvantages, this method must be resorted to whenever the occasion demands. The additional scarring, the lack of innervation and motion, and the difference in color index which result all must be overlooked.

New Drugs.—Widespread alterations in techniques have resulted from the advent of the antibiotics, cortisone, ACTH, heparin, and other drugs. The effectiveness of these drugs can be said to have shortened the treatment time and to have made the end result more successful.

There are widespread differences of opinion regarding the efficacy of cortisone and ACTH in the treatment of burns. Some authorities are just as opposed to their use as others are in favor of it. At this time it would seem that more evidence should be gathered before a decided stand can be taken as to the value of either cortisone or ACTH in the treatment of burns.

There is a modicum of evidence to make one believe that more research should be done before heparin is considered part of the armamentarium for healing wounds resulting from burns.

Much can be done to effect a quicker postoperative recovery by the addition of massage of burned tissues. This stimulation brings about early relaxation, enhanced blood supply, normal draping of tissue, destruction of fibrous connective tissue, and normal response on nerve impulse. Surely, this phase of treatment should not be overlooked.

Problem of Feeding.—The nutrition factor should have some consideration in the treatment of these patients. In most cases a bland, liquid, or semisoft diet will be in order. This can be taken by mouth, in some cases through a glass tube or straw. In the more severe cases, a Levine tube or indwelling catheter passed through the nose should be used. This type of feeding may help greatly in preventing many complications that might otherwise arise as a result of fluid imbalance, infection, or disturbance of grafts.

CONCLUSION

We contend that many mechanical, thermal, and electrical accidents can be prevented by better education of consumers, particularly in some of the simple principles of physics, mechanics, and chemistry. These principles should be taught to all high school students and to students in teacher-training institutions.

REFERENCES

1. Adams, F. H., Berglund, E., Balkin, S. G., and Chrisholm, T.: Pituitary Adrenocorticotropic Hormone in Severely Burned Children, J. A. M. A. **31**: 31-33, 1951.
2. Cole, W. H., and Puestow, C. B.: First Aid, Surgical and Medical, New York, 1942, D. Appleton Century Company.
3. Creditor, M., Bevans, M., Mundy, W., and Ragan, C.: Effects of ACTH on Wound Healing in Humans, Proc. Soc. Exper. Biol. & Med. **74**: 245, 1950.
4. Elrod, P. D., McCleery, R. S., and Ball, C. O. T.: An Experimental Study of the Effect of Heparin on Survival Time Following Lethal Burns, Surg. Gynec. & Obst. **92**: 35, 1951.
5. Howes, E. L., Plotz, C. M., Blunt, J. W., and Ragan, C.: Retardation of Wound Healing by Cortisone, Surgery **28**: 177, 1950.
6. Mote, J. R.: Proceedings of the First Clinical ACTH Conference, Philadelphia, 1950, The Blakiston Company.
7. Schultz, L. W., and Vazirani, S. J.: Electrical Burns of the Mouth, Am. J. Surg. **98**: 921-922, 1959.
8. Atterbury, R. A.: Antibiotics in Oral Infections, Fort. Rev. Chicago D. Soc. **24**: 9-13, 1952.

NASO-ALVEOLAR CYST

Report of a Case

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SWELLINGS of the upper lip present an interesting problem in differential diagnosis. A careful history and special roentgenographic techniques are often essential to a correct diagnosis. The particular area of the upper lip involved in the case to be presented could have been involved secondarily by pathoses arising in either the maxillary alveolar process, the nasal fossa, or the skin and subcutaneous tissues of the locality itself.

The developmental cyst in the case that follows was related to malfunction of specific embryologic developmental processes. The secondary inflammatory process which brought it to light resulted in a swelling which presented a characteristic deformity of the patient's face. The cyst was located entirely within the soft tissues of the face. The use of a contrast medium during the roentgenography was essential to this particular preoperative diagnosis.

CASE REPORT

A 32-year-old white man presented himself to the outpatient department of the 121 Evacuation Hospital (Korea) on July 21, 1955, complaining of a "painful and swollen face of three days' duration."

For an undetermined period ("more than three months") there had been a nontender, nonpainful, small enlargement of the patient's upper lip at the base of the right nostril. He sought no treatment for this swelling. During the past several months the patient had noticed a "small swelling" developing inside his right nostril at its lower border. On at least two occasions, this swelling "opened and a little fluid came out." After the drainage, the swelling inside the nostril and the lip "seemed to get a little better." During the past three days, the lip had become more swollen than it ever was before, and it was painful as well.

The past history and family history were essentially negative.

Examination.—On inspection, an enlargement of the right side of the patient's face could be discerned (Figs. 1 and 2) extending from the middle of the upper lip and the level of the right angle of the mouth superiorly to the level of the lower eyelid. The central focus of the swelling appeared to be the line angle formed by the junction of the alar cartilages forming the lateral border of the right nostril and the plane of the upper lip and face. The right nasolabial fold, which was well developed on the left side of the patient's face, was completely obliterated. The skin overlying the swelling was reddened.

On palpation, a fluctuant mass could be felt at the base of the lateral margin of the right nostril. It was tender to palpation. The extent of the mass could not be accurately determined.

On inspection, the floor of the right nostril was raised and appeared reddened and somewhat swollen.

Intraoral clinical and radiographic examination of all the patient's teeth and their surrounding structures revealed nothing of etiological significance. It was decided that, if possible, the mass should be aspirated to determine its contents and its configuration should be outlined with a contrast medium.

Fig. 1.

Fig. 2.

Fig. 1.—Preoperative view of secondarily infected developmental cyst. Note elevation of the right nostril.

Fig. 2.—Profile view showing extent of inflammatory process and obliteration of nasolabial skin fold.

On July 23, 1955, the mass was approached intraorally via the maxillary mucobuccal fold in the region of the upper right cuspid. After the region was anesthetized with 2 per cent procaine hydrochloride solution (with epinephrine 1:50,000), a 10 c.c. syringe with a 15-gauge needle was inserted into the fluctuant area. Approximately 3 c.c. of a cloudy, somewhat turbid, yellowish fluid was removed; this was immediately replaced by a similar amount of Lipiodol contrast medium, and posteroanterior and lateral roentgenograms of the head were taken (Figs. 3 and 4). The roentgenograms revealed a well-defined, oval cystic cavity situated in the soft tissues of the face at the junction of the upper lip and the lateral wall of the right nostril. The cystic cavity was disclosed to be entirely within the soft tissues and lying very close to the anterior surface of the maxilla.

Physical Examination.—The patient, a well-developed, well-nourished white man in no acute distress, was alert and cooperative. No other pertinent findings were revealed by a general physical examination.

Complete blood count and urinalysis were within normal limits. Serologic tests for syphilis were negative.

Treatment.—Upon presentation the patient was given procaine penicillin, 600,000 units, intramuscularly. This was continued daily until the fifth postoperative day, a total of eight days.

On July 25, 1955, the cystic mass was excised. An intraoral avenue of approach by way of the upper mucobuccal fold was selected. The tissues of the maxilla were anesthetized locally by infiltration injection of 2 per cent procaine hydrochloride (with epinephrine 1:50,000). The tissues were infiltrated from the region of the maxillary labial frenum posteriorly to the region of the upper right second molar. The anesthetic solution

Fig. 3.—Lateral roentgenogram of skull showing cyst filled with contrast medium. Note that the lesion is entirely within the soft tissues.

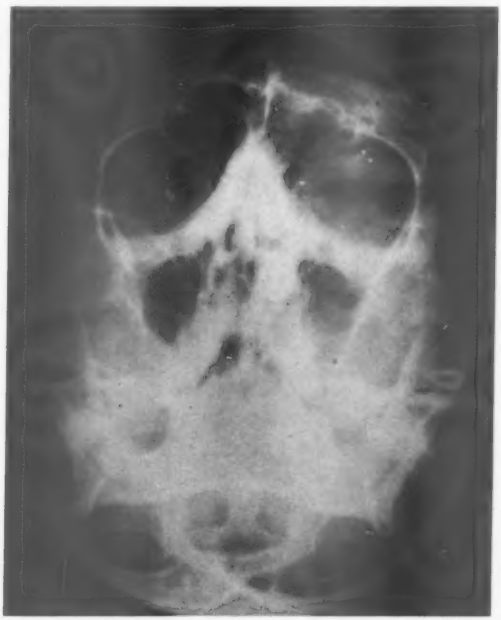

Fig. 4.—Posteroanterior view of the skull as viewed from the posterior aspect. Note the location of the cyst.

was also deposited by direct external approach in the region of the infraorbital foramen and canal. The external approach was selected to avoid encountering the cyst, which was situated near the path of the usual intraoral approach to the infraorbital foramen. The ala of the right nostril was infiltrated from below via the mucobuccal fold and interiorly through the nostril itself.

An incision was made in the upper mucobuccal fold, from the labial frenum posteriorly to the region of the upper right second molar. The incision was made through the mucosa and submucosa only. The deeper muscle fibers were dissected bluntly with small curved and straight Kelly mosquito forceps.

The cystic sac was found to be readily recognizable and quite fibrous and tough. It was well defined on all sides except superiorly in the region of the nostril. Blunt dissection was sufficient for the isolation of the cyst on all sides except the above-mentioned superior aspect where it was attached to the overlying skin. This was evidently the site of the fistulous tract that formed and provided drainage previously.

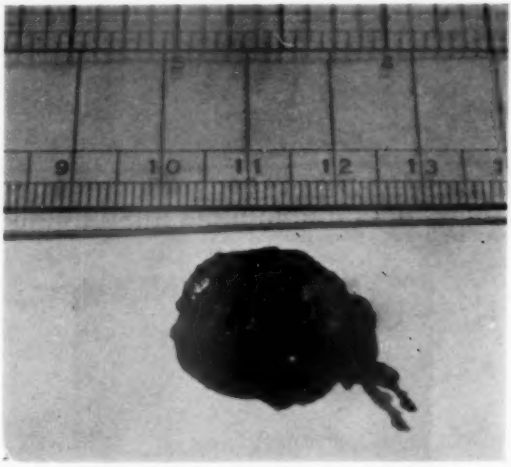

Fig. 5.—View of the specimen immediately after removal. Note, at the right, the remains of the sinus through which drainage occurred via the right nostril.

In this region the tract and its overlying skin inside the nostril were excised by sharp dissection. The cyst, its fistulous tract, and overlying attachments were removed in one piece (Fig. 5). The deep portion of the wound was closed with interrupted 000 chromic gut sutures. Intraorally, the mucosa was closed with interrupted 000 black silk sutures. The line of incision in the right nostril was closed with one 000 black silk suture. Hemorrhage was controlled by external pressure. Ice packs were applied to the patient's face twenty minutes every hour intermittently for the first twelve postoperative hours. Pain was controled adequately with 5 gr. A.P.C. with ½ gr. codeine sulfate every four hours as needed. The patient was placed on a liquid diet. On the third day, soft foods were added to the diet. On the fifth day, the sutures were removed. On the sixth day, the patient returned to a normal diet. Thereafter, the wound healed uneventfully.

Pathologist's Report.—The section showed a cyst wall made up of rather edematous connective tissue markedly infiltrated by plasma cells and lymphocytes and in areas intermingled with numerous pigment-containing macrophages. The inner surface of this wall was lined partly with multilayered squamous epithelium and in some areas with ciliated epithelium (Fig. 6). Rete pegs were absent.

Diagnosis.—Cyst, secondarily inflamed, of developmental origin.

The patient was seen periodically for the next three months, and at the end of that time the area from which the cyst was removed had healed completely and uneventfully (Fig. 7).

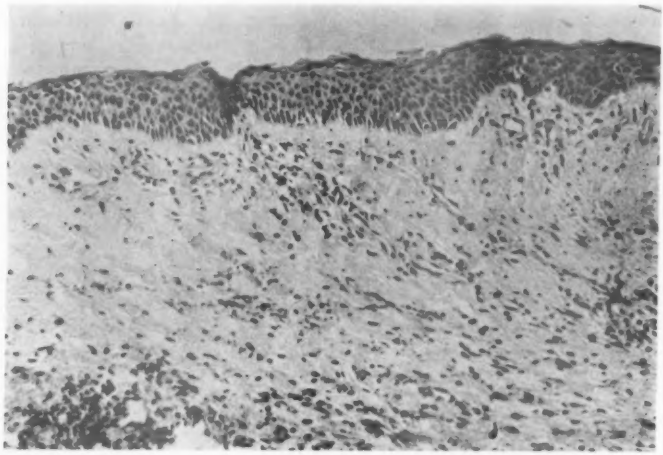

Fig. 6.—Photomicrograph of the specimen. Note the fibrous capsule and the stratified squamous epithelium lining. Note also the complete absence of rete pegs.

Fig. 7.—Photograph of patient one month after surgery. Note return of symmetry and the nasolabial skin fold.

DISCUSSION

The embryological development of the face is characterized by the fusion of various soft-tissue processes that develop surrounding the primordial stomatodeum. Often the epithelium covering the processes prior to fusion is not entirely

eliminated and remains instead as atrophic rests in the substance of the area of fusion. These epithelial rests can, with stimulation, proliferate, and lead to cyst formation. The so-called developmental cysts are found, therefore, in specific and characteristic sites.

The naso-alveolar cyst is closely related to the globulomaxillary cyst. The former represents a more superficial and the latter a deeper evidence of epithelial rest proliferation along the same line of fusion. The kind of cyst presented in this case, which lies entirely within the soft tissues, is the least commonly encountered; however, it produces the most characteristic facial deformity when enlarged.

A TECHNIQUE FOR SIALOGRAPHY

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IN THIS article a method of using a dental cartridge syringe to produce sialograms is described. This instrument has been found more convenient for the purpose than syringes previously used.

Much of the difficulty in making sialograms stems from the narrowness of the opening leading into the ducts, particularly the submandibular duct. Also, an actively secreting salivary gland will rapidly empty itself of any contrast medium that is introduced.

The problem produced by the narrow entrance may be approached in a number of ways. Sialogogues may be used, so that the duct opening is distended by saliva and is easily seen. This undoubtedly makes cannulation easier, although an efficient sucker is required for the patient's comfort, to prevent swallowing, and to maintain optimum visibility. Unfortunately, the injection of constrast medium against a fast salivary flow can cause sufficient distention of the gland to be unpleasant for the patient. Alternatively, the duct entrance may be dilated with lachrimal duct dilators. This is not always easy and may require the use of a local anesthetic. Finally, a cannula which is fine enough to pass the narrow opening without its being dilated may be used.

In order to have sufficient contrast medium in the ducts at the time the radiogram is taken, the gland can be overfilled so that, even though some is rejected, enough remains to produce a satisfactory picture. This technique has two disadvantages: (1) Overdistention of the ducts can cause pain and (2) the contrast medium which refluxes into the mouth can spoil the radiogram. The alternative is to close the duct orifice in some manner so as to retain the fluid. Whichever method is used, a viscous contrast medium will help to delay emptying.

A viscous medium will also push its way into the duct better than a fluid one. This is an advantage in that it opens up the duct in front of the cannula. On the other hand, if a narrow-gauge cannula is chosen, the viscous contrast medium will be difficult to inject. A small-diameter syringe and plunger will be required to produce the necessary pressure for this combination, and the cannula will have to be firmly fixed to the barrel of the syringe or it may be pushed off during the injection.

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EQUIPMENT

Dental cartridge syringe needles are made in narrow gauges; they can be readily turned into strong, flexible cannulae; and they are attached firmly to the syringe by a screw hub. The cartridge syringe itself is designed to withstand the injection of liquid under moderate pressure, and, it has the necessary small-diameter plunger to make this easy. The type fitted with a comfortable crutch handle is very suitable for sialography.

The cannulae used in this technique are modeled after those described by Rose[2, 3] and by Ollerenshaw and Rose.[1] For parotid sialography 23 gauge, 42 mm. cartridge needles are used, and for submandibular sialography either 23 or 26 gauge, 42 mm. needles may be required.

With the stylet in place to protect the lumen, the point of the needle is ground away and the end carefully rounded and polished. A bend is made to form an angle of about 140 degrees 6 mm. from the tip for a parotid cannula and about 2.5 cm. from the tip for a submandibular one. A blob of cold-cure acrylic is added; this will occlude the entrance to the duct and prevent reflux during and after the injection. First the hub is threaded on; then a small amount of freshly mixed acrylic is molded to form a fusiform blob. This should be placed a clear 3 mm. from the end for a parotid cannula and 2 cm. from the end for a submandibular one. To settle the acrylic into firm contact with the metal, an additional drop of monomer is added to the acrylic and the cannula is vibrated with the handle of a Le Cron carver. Further powder may be dusted on to mop up the excess liquid. When the blob has hardened, it may be trimmed and polished, after which the stylet can be removed, and the cannula is ready for use.

At the present time, cartridges of contrast medium are not available commercially. Immediately before the sialogram is to be taken, therefore, a cartridge of local anesthetic should be sterilized by boiling. While it is held in a sterile swab, the plunger end is removed with a hooked probe and the local anesthetic is emptied out. Viscous contrast medium, previously drawn up into a sterile 5 c.c. syringe, is injected into the cartridge through a large-bore needle and the plunger is replaced. Any air trapped by this maneuver can be released by passing a probe down by the side of the plunger.

Great care must be taken that the cartridge is only filled immediately before use, so that there is no chance of its being mistaken for a cartridge of local anesthetic. As soon as it has been used, it should be destroyed.

Additional items of equipment required are a saliva ejector, some gauze swabs, half a cotton-wool roll, and a towel to protect the patient's clothing. Submandibular sialography is best carried out at the dental chair by means of a dental x-ray machine. Parotid gland sialography, on the other hand, should be done on the radiographic table or with the patient positioned against the Potter-Bucky diaphragm of a sinus stand using a full-sized x-ray set.

THE TECHNIQUE OF SUBMANDIBULAR SIALOGRAPHY

The submandibular duct is a thin-walled tube which measures up to 0.5 cm. in diameter, but its entrance at the sublingual papilla may be minute. From the papilla the duct runs downward and backward on the lingual side of the sublingual fold, diverging from it at an acute angle.

In order that the opening may be found, the floor of the mouth should be dried and the papilla watched until it is seen opening and closing as the saliva escapes. If necessary, the gland may be massaged to express a little saliva. The position of the entrance is variable; it may be at the tip of the papilla, just short of the tip on its superior aspect, more commonly to the posterior of the tip, or even under the papilla.

The cartridge syringe is loaded with a cartridge of viscous contrast medium, and a small amount is expressed to fill the cannula and prove its patency. Usually the 26 gauge cannula is required, although sometimes the entrance is large enough to take a 23 gauge one.

The patient is asked to curl his tongue up and open his mouth. After a saliva ejector has been placed on the opposite side, the cannula is introduced into the duct from the same side as the gland to be examined (Fig. 1, *A*). As it enters, the syringe handle is swung upward toward the midline and then downward toward the opposite side (Fig. 1, *B*). This brings the cannula into line with the main part of the duct. Sometimes the cannula enters the narrow entrance but does not slip onward into the main duct. If this happens, the injection of a small amount of fluid will dilate and lubricate the duct ahead of the cannula and allow it to slip in until the acrylic blob plugs the entrance.

Once the entrance has been negotiated, it is important that the cannula be directed backward and downward, as in a number of patients the duct of Bartholin's sublingual gland enters Wharton's duct near the papilla. If the cannula is directed along the line of the sublingual fold, it will tend to enter this duct. Should this happen, the cannula will be arrested after only 1 cm. has been introduced and distention of the sublingual fold will be seen as soon as the injection is started. Bartholin's sublingual gland will take only about a 0.25 ml. of fluid, and if more is injected the patient will complain of pain. Provided the injection is stopped at this stage, no harm will be done and the cannula can be withdrawn until the tip is just inside the papilla. As some of the fluid will tend to reflux into Wharton's duct, the cannula can now be made to enter it if directed in the appropriate direction.

Once the cannula is properly positioned, the injection can begin. Although a moderate pressure is required to force the viscous fluid through the fine cannula, the contrast medium will escape into the duct as only a slow trickle. This is an advantage, as it allows the gland time to accommodate itself to the process. Filling the submandibular duct system requires between 0.5 and 1.0 ml. of contrast medium. At the end of the injection the gland will form an easily visible and palpable swelling in the submandibular triangle (Fig. 1, *D*), and the patient will experience a sensation of tightness. Distention

of the sublingual fold toward the *end* of the injection indicates the filling of Bartholin's gland by reflux.

Submandibular sialography performed in this manner should not require the use of even a topical anesthetic and should cause only minimal discomfort. If the patient complains of pain, it is a good idea to stop the injection and take a radiogram to find out what is happening. Gross overfilling of the gland

Fig. 1.—*A*, The tip of the cannula is touching the entrance to the right submandibular duct. Note the direction in which the cannula enters the duct.

B, The syringe has been swung upward, forward, and downward and now lies in the duct.

C, The injection has been completed, the syringe unscrewed from the hub and half a cotton-wool roll impaled on the end of the cannula. In this figure the cannula is seen preventing reflux from the duct.

D, The characteristic swelling in the submandibular triangle is seen following a successful injection. The end of the cannula is held between the lips.

may cause pain, as will the distention of a duct completely blocked by some obstruction. The passage of contrast medium past a neoplasm ulcerating into the duct is also painful. In all these cases the fluid already injected by the time the pain is felt will usually be sufficient to make a radiographic diagnosis possible.

Provided no force is used in introducing the cannula, penetration of the wall of the duct should not occur. As most of the difficulties in cannulating the duct arise in negotiating the entrance, a perforation will probably occur near this point. Contrast medium will then appear under the mucous membrane of the floor of the mouth as soon as the injection is started and the patient will complain of pain. Most of the contrast media are relatively innocuous and, should such an accident happen, small amounts are unlikely to cause trouble.

As soon as the injection has been completed, the needle hub is unscrewed and the cannula is grasped with forceps and withdrawn from the syringe. Half a cotton-wool roll is impaled on the sharp, reverse end of the cannula, which is left in situ to close the duct while the radiograms are taken (Fig. 1, *C*).

Any saliva or spilled contrast medium is aspirated from the floor of the mouth and the saliva ejector is removed.

Radiography for Submandibular Sialography.—Plain films should always be taken before sialography is started, in case a calculus should be present in the duct. By this routine, positioning of the patient is also practiced and exposure times are checked, reducing the possibility of an error in technique when the sialographic films are taken. The views required are a central true occlusal view of the floor of the mouth, a posterior oblique occlusal view,[5] and an oblique lateral jaw film.

A sialogram is a valuable diagnostic aid if a stone is present in the posterior part of the duct, but it should not be performed if a stone lies anteriorly lest the procedure displace it backward. Even if films taken on a previous occasion show a stone in the posterior region, further plain films are desirable as the stone may have moved forward in the interval.

Following the injection, an oblique lateral jaw film and a posterior oblique occlusal film should be taken. For the oblique lateral view, fast nonscreen films are preferred, and the face is positioned higher up the film than if the jaw itself were to be radiographed. An angle of tilt on a vertical angle board of 25 degrees is satisfactory, with the same rotation of the head as would be used for radiographing the lower third molar. When a small-diameter cylinder cone and a 14 inch anode-film distance are used, the exposure is about 0.75 second at 15 Ma. and 77 kv. (Fig. 2).

If possible, the posterior oblique occlusal film is taken with the cannula still in place, but if papillary stenosis is suspected another film should be taken after the cannula has been removed (Fig. 3). Normally, most of the contrast medium is ejected by the unobstructed gland within a few minutes, but when sialoangiectasis is present some may be retained for hours or even

days. An additional film taken after twenty minutes as an "excretion" sialogram will then clearly define the diseased and nonfunctioning segments of gland. The position of a small obstruction may sometimes be more clearly defined by this method. Provided no major obstruction exists, a sialogogue may be given to speed up the emptying process and a secretion sialogram may be performed.[4]

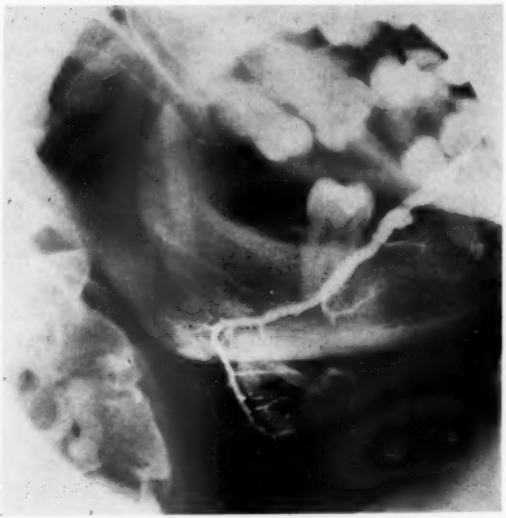

Fig. 2.—A typical oblique lateral view of a left submandibular sialogram. Note complete filling of all the small ducts and faint but distinct outlining of the gland lobules. Two posterior sublingual ducts are seen joining Wharton's duct. The more posterior of these appears to replace the deep part of the submandibular gland.

Fig. 3.—The posterior oblique occlusal view of a submandibular sialogram. Again a posterior sublingual duct joins Wharton's duct. Note that as the cannula has been removed contrast medium is already returning from the duct.

Following a normal examination, the gland may remain somewhat swollen for an hour or two, even though all the contrast medium has been returned. The patient should be told of this possibility and suitably reassured.

After a successful injection, the radiograms should show the duct tree well filled with contrast medium. All the fine branches should be visible, but only a faint outline of the gland lobules should be present. The injection of sufficient dye to produce heavy lobular shadows is painful for the patient and unnecessary from a diagnostic point of view.

THE TECHNIQUE OF PAROTID SIALOGRAPHY

The parotid duct opens at the tip of the parotid papilla, which is to be found on the buccal mucosa opposite the second upper molar. From the entrance the parotid duct runs backward through the cheek. The opening is wider than that of the submandibular duct, but the duct itself is narrower. For the first part of its course, it runs backward and submucosally for 3 to 5 mm. It then turns laterally, passing through the buccinator muscle, and across the anterior border of masseter muscle. Finally, it inclines backward, downward, and then medially. During the intraglandular part of its course, large branches are given off, first on one side and then on the other.

As Ollerenshaw and Rose[1] have pointed out, the cannula should enter the submucosal part of the duct only. The blob of acrylic should both block the entrance and prevent further passage of the cannula into the duct beyond the point where its tip lies just short of the first bend. The cannula described earlier has the blob of acrylic 3 mm. from the tip, so that it will fit the duct in the manner just described. As the parotid duct entrance is larger than that of Wharton's duct, a 23 gauge cannula can be used.

A syringe is assembled and loaded with a cartridge of contrast medium in a manner similar to that described for submandibular sialography.

The duct orifice is usually obvious (Fig. 4, *A*); however, should this not be the case, a little saliva may be milked to the entrance by stroking the cheek. Once the entrance is identified, there is usually no difficulty in inserting the cannula (Fig. 4, *B*). Next, 1 to 2 ml. of viscous contrast medium is slowly injected. Guides to the amount to be used are visible swelling of the parotid region (Fig. 4, *C*), the sensation of tightness experienced by the patient, and the resistance felt to injection. As soon as the injection is completed, the cannula is removed and the duct entrance is compressed against a swab with the finger and thumb (Fig. 4, *D*).

It is not as easy in parotid sialography to occlude the duct opening during radiography as it is during examination of the submandibular gland. However, satisfactory radiographs may be obtained if the duct is compressed as described, both before the films are taken and between exposures. Papillary stenosis is a more common cause of obstruction to the parotid duct than to the submandibular duct, and the narrowed terminal segment found in this condition may be masked if the cannula is left in position. This being so, the method described is not without its advantages. Of course, it is not possible to compress the submandibular duct adequately in this manner.

Fig. 4.—*A*, A parotid cannula points out the entrance to the right parotid duct.

B, The cannula is seen in place in the duct with the acrylic blob occluding the entrance.

C, Photograph showing the characteristic swelling of the parotid gland after a satisfactory injection.

D, The method of holding the papilla between the finger and a swab inside the mouth and the thumb outside.

Radiography for Parotid Sialograms.—General radiographic equipment is required, and as many preparations as possible should be completed before the injection is made, so that the minimum time elapses between the completion of the injection and the taking of the films.

A.

B. *C.*

Fig. 5.—*A*, Off-center anteroposterior view of a left parotid sialogram. Note again the correct degree of filling. A small radiolucent obstruction is indicated by the arrow.

B, A lateral view of the same patient. Again the radiolucent obstruction is seen. The tube has been angulated a little toward the spine to demonstrate the posterior part of the gland.

C, The oblique lateral view of the same patient. Here the parotid gland is viewed obliquely from below, but the outlines of the lobules are more easily seen. Once more the obstruction is observed. An additional film taken after fifteen minutes showed the duct empty anterior to this obstruction but still filled behind it. The gland slowly emptied over the next few hours.

The views taken are an off-center anteroposterior (Fig. 5, *A*), a true lateral (Fig. 5, *B*) and an oblique lateral jaw (Fig. 5, *C*). The off-center anteroposterior film is taken at 4 feet, and a Potter-Bucky diaphragm is required. The back of the patient's head is placed against the table, with both the sagittal plane and the line joining the outer canthus of the eye and the tragus of the ear at right angles to its surface. The tube is directed perpendicular to the film at the level of the tip of the nose, but offset 1 inch from the sagittal plane toward the side to be examined. About 75 kv. should be used for the average adult.

For the lateral view, the Potter-Bucky diaphragm is again used and the patient is positioned as for a lateral sinus view. If the patient has teeth, the jaws should be propped a little apart with a radiolucent block or pad of gauze placed between the incisor teeth. It is important to extend the head on the neck, so that the cervical spine is kept as clear as possible of the parotid gland. About 68 kv. should be used to give a low-contrast film; otherwise, the bones will overshadow the duct tree. Should an important shadow be partially obscured by the spine, an additional film can be taken, with the tube angulated a little toward the spine.

For the oblique lateral view, the patient's head is placed so that the sagittal plane is parallel with the film. The chin is thrust forward and the head is extended on the cervical spine. The tube is tilted toward the head through an angle of 30 degrees and centered below the opposite ramus of the mandible. For this view, one should use 65 kv. with screen-sensitive films in a cassette or 77 kv. with nonscreen film.

As with films of the submandibular gland, the films should show the ducts well filled but with a minimum of lobular filling. Again, if required, "excretion" or "secretion" films may be taken after twenty to thirty minutes.

My thanks are due Messrs. A. G. Allen, G. T. Hankey, W. R. Keizer, S. G. Allen, and Professor A. E. W. Miles, Consultant Dental Surgeons, and Mr. W. T. Irvine, Reader in Surgery, for permitting me to do the sialography for their patients. I am also grateful to Mr. G. T. Hankey, Lecturer in Dental Radiology, for permission to use the radiographs and Mr. P. Broadberry for copying them.

REFERENCES

1. Ollerenshaw, R., and Rose, S.: Sialography—a Valuable Diagnostic Method, D. Radiog. & Photog. **29**: 37, 1956.
2. Rose, S. S.: Sialography in Diagnosis, Postgrad. Med. **26**: 521, 1950.
3. Rose, S. S.: A Clinical and Radiological Survey of 192 Cases of Recurrent Swellings of the Salivary Glands, Ann. Roy. Coll. Surgeons England **15**: 374, 1954.
4. Rubin, P., and Holt, J. F.: Secretory Sialography in Diseases of the Major Salivary Glands, Am. J. Roentgenol. **77**: 575, 1957.
5. Seward, G. R.: Notes on Dental Radiography. II. Bite-Wings and Occlusals, D. Practitioner **4**: 250, 1954.

ORAL MEDICINE

THE ORAL LESIONS OF LICHEN PLANUS

Observations on 100 Cases

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LICHEN PLANUS, or lichen ruber planus, is a relatively common inflammatory disease of the skin and mucous membrane. The mucosal surface most frequently involved is that of the oral cavity. Because of the considerable variability in the clinical appearance of the oral lesions of lichen planus, particularly in the different areas of the mouth, a reconsideration of this clinical entity was felt to be of interest. Since many textbooks describe only "lacelike" lesions of the buccal mucosa, there has been a tendency to recognize only this form of lichen planus.

HISTORICAL REVIEW

The disease was first described in 1869 by Erasmus Wilson[37] as "leichen planus," "an eruption of pimples remarkable for their colour, their figure, their structure, their habits of isolated and aggregated development, their habitat, their local and chronic character, and for the melasmic stains which they leave behind them when they disappear." Among the numerous cases in Wilson's detailed study, eruptions are noted on the buccal mucosa, tongue, and pharynx. The oral lesions in the case of a 57-year-old woman are described as an eruption presenting the appearance of white specks on the side of the tongue. The round white spots on the tongue, buccal mucosa, and lower lip of a 56-year-old woman are described as comparable to the skin papules but lacking prominence.

The oral lesions in lichen planus were further noted and described by Unna[35] and Crocker,[8] the latter noting white lines and white spots on the buccal mucosa and symmetric plaques on the sides of the tongue in several cases. Thibierge[33] first described the oral lesions systematically. He felt that in most cases the lesions occurred on the buccal mucosa and the tongue, with certain differences

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in appearance. On the tongue the eruption was described as white spots, round or slightly irregular in shape, and without notable prominence. The lingual papillae appeared diminished in depth. The spots might be isolated or joined, and in some cases they formed parallel lines at the border of the tongue. On the buccal mucosa the lesions were usually numerous small papules, rounded or stellate in shape, pure white, and often shiny. The papules were isolated and formed either plaques or interlacing, diversely placed lines. Between papules, the mucosa presented superficial ulceration. Although the plaques could cover the entire buccal surface, they were usually localized to the area of the molars. In the rest of the mouth the papules were isolated or present in small numbers.

At this time it was felt that the oral lesions were merely an accompaniment to the generalized skin eruption. It was Audry[2] who pointed out that the oral lesions of lichen planus could occur in the absence of skin lesions. Poor[29] first described the formation of cavities in lichen planus of the mucosa, corresponding in character to subepithelial bullae and characterized by exudation from surrounding blood vessels. Dubreuilh[10] stated that, aside from the very numerous cases of lichen planus involving both skin and mucosa, the involvement of oral mucous membrane alone was more common than involvement of the skin without mucosal lesions. From a study of biopsy specimens from lesions of the tongue and buccal mucosa, he felt that histologically these lesions were comparable in all points to those of the skin. Furthermore, because of the ease of oral biopsy, Dubreuilh suggested this as a diagnostic aid. The principal lesions were described as a cellular infiltration into the connective tissue consisting of lymphocytes with rare polymorphonuclear leukocytes and plasma cells. In the tongue the infiltration was most marked between papillae. The epithelium was described as showing only some hyperplasia and edema. Lieberthal,[20] who first described the oral manifestations of lichen planus in the American literature, realized that the lesions of the tongue differed from those of the buccal mucosa. The tongue usually had irregular oval or circular plaques, whereas the cheek usually had papules, oval or circular plaques, most frequently streaks or crisscrossing linear projections. The tongue lesions were described as smoother, less hard, not raised above the level of the normal surface, and less glossy than the buccal lesions. Thus the pertinent facts concerning lichen planus had been set down with considerable clarity by 1907.

Gottfried Trautmann,[34] in addition to describing lichen planus bullosa, made a study of the medical literature up to 1910 and outlined a preliminary survey of the occurrence and disposition of oral mucosal lesions in lichen planus. Oral lesions associated with skin lesions were observed in ninety-four out of 157 cases of this dermatosis. In twenty-six cases the oral lesions were confined to the oral mucosa with the cheek (82 per cent) and tongue (50 per cent) being most commonly involved. Lesions of the lips (22 per cent), palate (17 per cent), and gingiva (10 per cent) were less commonly seen.

Since Trautmann's survey, few such studies have been carried out. Culver[9] found involvement of the oral mucosa in thirty-one of 148 cases of lichen planus studied. Of these, twenty-three had lesions on the buccal mucosa and six had

lingual involvement. Little[21] reported mucous membrane lesions in forty-eight of 270 cases of lichen planus. Lichen planus has been reported to affect male and female patients in about equal numbers. Some authors have reported a greater number of males affected,[9] while others have considered the female to be more often affected.[21] Lichen planus commonly affects the middle-aged person but cases have been reported in children.[3] The oral mucosal lesions can occur at any age.

At present the cause of lichen planus is not known, although psychosomatic and hereditary factors appear to be of some importance. The numerous theories advanced in the past to explain the etiology of the disease may be listed as (1) traumatic, (2) specific bacterial, (3) syphilitic, (4) parasitic, (5) viral, (6) mycotic, (7) allergic, (8) toxic, (9) nervous, (10) hereditary, and (11) psychosomatic. Cases of lichen planus with oral manifestations have been reported in several members of the same family,[5] and Epstein[11] has described lesions confined to the oral cavity in twins. Reported isolation of specific microorganisms from lichen planus lesions has not been confirmed. Lichen planus has been reported to occur following the use of certain drugs, such as gold and arsenic.[15] Recently, the antimalarial drug, Atabrine, has been reported to result in a dermatitis with oral manifestations closely resembling lichen planus.[4]

PRESENT STUDY

Our discussion of lichen planus is based upon information and impressions gained from 100 oral cases. These cases were seen by us in private practice, in consultation, and in the oral medicine clinic at Tufts University School of Dental Medicine. There were forty-four female and fifty-six male patients, ranging in age from 13 to 78 years. Over 50 per cent of these patients had skin lesions as well as oral lesions, and many of the cases originally confined to the oral mucosa, have presented skin lesions as the disease progressed. Many patients originally presenting with skin involvement later developed oral lesions. It is our impression, based on clinical evaluation of our series of patients, that most cases of lichen planus will eventually show both skin and oral mucosal lesions. It has been our experience that if the patients are followed for a sufficient length of time, and if the examination of the skin and mucous membrane is thorough, both cutaneous and mucosal involvement will be seen in most patients; either kind may appear first. We have gained this impression gradually over the past ten years, and in many cases we originally classified the condition as oral, skin, or a combination of both. For this reason, we will not attempt to present exact statistics as to the percentage of lichen planus cases confined to the skin or to the oral mucosa. Our cases have been followed for varying periods up to ten years. Through the courtesy of Professor Francis McCarthy, we were able to evaluate and study some cases originally seen by him at Tufts University and in private practice.

As to disposition of the oral lesions in our series, approximately 80 per cent of the patients had buccal lesions and 65 per cent had lingual involvement. Labial involvement occurred in 20 per cent of the cases. Lesions of the gingiva,

floor of the mouth, and palate were rarely seen (less than 10 per cent). A larger series of cases, followed for a longer time, would give us more accurate figures.

A general observation may be made in relation to etiological factors. It has been our impression, based on experience with a large series of cases, that lichen planus represents a reaction of the skin and mucous membrane to some form of emotional trauma. Many of our cases have occurred in connection with a significant and severe psychological stress, such as the death of a close friend or relative, marital and sexual problems, failure in a career, or loss of job and security. We have seen several cases in medical and dental students prior to final examinations. We have noticed that frequently the particular incident may have reached a conclusion before the first symptoms of lichen planus appeared. We realize that this is a difficult hypothesis to prove, but we feel that the frequent association is more than coincidence. Further evidence to support this concept is the absence of lichen planus among psychotic persons. One of us (P. L. M.), as consultant to a large mental hospital, has not encountered lichen planus among any of the many psychotic patients referred because of dermatologic problems. Several observers have made this same observation with respect to other "psychosomatic diseases." Further investigation of the possible psychosomatic etiology of lichen planus is indicated. Psychiatric evaluation of lichen planus cases may reveal significant information.

SYMPTOMS

Symptoms are usually absent in relation to oral lesions, although on the skin there tends to be a severe pruritus. In the oral cavity there may be mild subjective symptoms, usually described as a "burning" or "irritation." In rare cases a "burning tongue" may signify lichen planus rather than the usual papillitis and glossitis.

The lesions are usually discovered accidentally when the patient feels them with his tongue or sees them in a mirror, or when they are noticed by the dentist or physician.

If the lesions are erosive or bullous in nature, symptoms may be very severe. Some of the patients experience extreme pain from the eroded areas and are as uncomfortable as those suffering from such bullous eruptions as pemphigus or erythema multiforme.

In the female patient, soreness and irritation of the mucosal lesions may be more pronounced at the time of the menses.

CLINICAL APPEARANCE OF THE LESIONS

The fundamental lesion of lichen planus is a small papule. This "eruptivelement"[34] of the European investigators is the size of a pinhead, domed or hemispherical in configuration, and glistening white. The term "mother-of-pearl white" has been utilized in the literature to emphasize the highly polished appearance of the surface.[34] These papules may be somewhat flattened, and the

color may vary from white to gray. On the tongue the lichen papules present some variation from those on the rest of the oral mucosa. They tend to be more flattened, and they do not glisten.

The appearance of lichen planus lesions depends upon the various arrangements of the minute papules. They may remain as isolated pinhead lesions on a reddened base, or they may coalesce and arrange themselves in the form of lines, circles, dense plaques, and other configurations. These figures are not specific types of lesions, and numerous variations of the pattern may exist in the same case. For this reason, we feel that classification of the types of lesion is of little value except for descriptive purposes.

Fig. 1.—Lichen planus eruption on the flexor surface of the right arm in a 40-year-old man. Note the numerous small confluent papules forming plaques. Note also the linear pattern at the folds of the skin (left).

The grayish white lines and dots representing the arrangement of papules were described as a pathognomonic sign by Wickham[36] in 1895. Fox[12] described the oral lesions as minute, punctate, linear streaks, circles, reticulated areas, or solid areas. Gougerot[13] described reticular, annular, plaque lesions as well as ulcerated, dotted, and sclerotic forms. Cooke,[7] in his recent survey of fifty oral cases, has classified the patterns into different groups. A *linear* pattern is often seen on the buccal mucosa; a *discrete papular* pattern is commonly seen on the buccal mucosa; a *reticular* pattern is often seen on the buccal mucosa and is most frequent on the gingiva; an *annular* pattern is noted occasionally; a *pigmented* pattern may occur on the soft palate; and *vesicular* or *bullous* lichen planus and *atrophic* or *erosive* lichen planus are also discussed.

We have found that different areas of the oral mucosa appear to form certain lesion patterns, and we shall discuss the clinical appearance of oral lichen planus with reference to the sites involved.

BUCCAL MUCOSA

The buccal mucosa is the oral site most commonly involved. In approximately 85 per cent of our cases there was buccal mucosal involvement. When the lesions were small, they tended to be placed posteriorly and at the

Fig. 2.

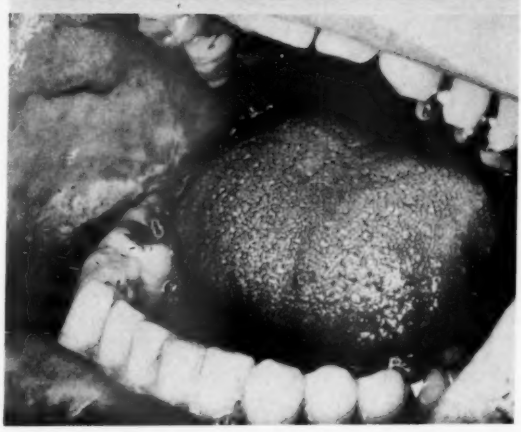

Fig. 3.

Fig. 2.—Lesions on buccal mucosa. Reticular or typical lacelike patterns involving a large area on both right and left buccal regions. These were the initial lesions in a 22-year-old man; no skin involvement has followed to date (a period of eighteen months).

Fig. 3.—Plaque pattern at interdental line, bilateral, in a 35-year-old man. Skin lesions were also present.

level of the occlusal or interdental line (Fig. 3). Occasionally a prominent hyperkeratotic occlusal line may be mistaken for a linear lichen planus involvement. The buccal mucosa also harbors the greatest variety of lichen planus lesions.

Reticular pattern: This is the most common pattern on the buccal mucosa. Lesions may be localized to the area of the interdental line or involve the entire buccal mucosa, extending to the vestibular and retromolar areas. The pattern is one of interlacing raised lines forming a latticework (Fig. 2).

Punctate linear pattern: Individual papules may appear as a series or mass of white dots on a violaceous background. The papules may arrange themselves into a series of more or less parallel lines running in a direction similar to the interdental line.

Plaque pattern: This is usually a fairly solid, raised, white plaque. Closer inspection with the naked eye or with a magnifying glass reveals the plaque to be composed of large numbers of minute papules (common on the tongue).

Papular pattern: In this type, the papules are spaced somewhat apart and yet close enough that the lesion has a pebbled white or gray appearance. This is somewhat similar to the plaque type of lesion, but the individual nature of the papules is much more obvious in this form.

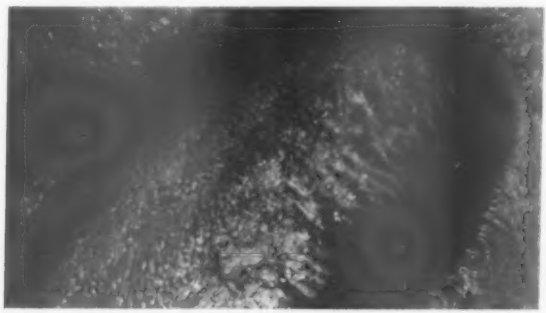

Fig. 4.—Lingual lesions, reticular pattern. Note the striations along the lateral border of the tongue.

Erosive or ulcerative pattern: Lesions of lichen planus rarely ulcerate, and we have seen very few on the buccal mucosa. When they occur, they are usually noted at the line of occlusion. Ulcerative lesions appear to be related to traumatic influences or local irritation. Milian and Fouquet[25] reported buccal ulcerations 1 cm. in diameter surrounded by typical papules. Other cases of buccal ulcerative lesions were noted by Lebourg[18] and others. Ulcerative lesions are not necessarily preceded by vesicular lesions.

Vesicular pattern: Vesicular or bullous lesions of lichen planus are rare on the buccal mucosa, but they do occur.[1] These lesions will be discussed in more detail in relation to lingual involvement.

TONGUE

Lesions on the tongue differ from those on the buccal mucosa in many features, and there is again considerable variability in the appearance of the lesions.

Reticular pattern: Interlacing, sharp, white striae are the typical pattern; these lesions are often seen on the lateral borders of the tongue (Fig. 4), but the entire dorsal surface may be covered (Fig. 5). This type of involvement has a tendency toward bilateral distribution. While the lateral borders of the tongue are involved, the dorsum may be completely free of lichen planus lesions. Lingual papillae atrophy in areas of involvement, and this smooth surface accentuates the white striae.

Fig. 5.

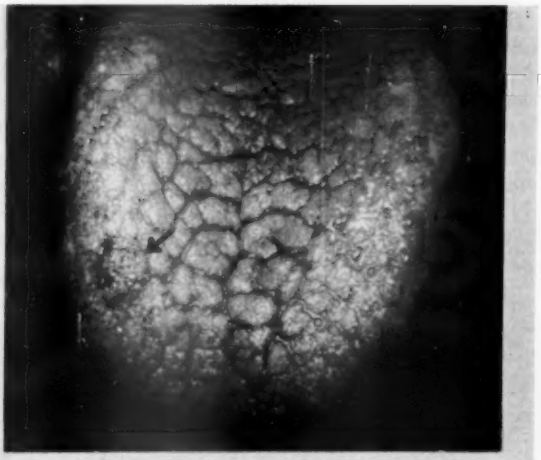

Fig. 6.

Fig. 5.—Lingual lesions with generalized reticular and verrucous pattern in a 45-year-old woman. Note the raised circumscribed area at left and the generalized white latticelike pattern. The congenital fissuring is an incidental finding.

Fig. 6.—Lingual lesions, annular pattern, in a 21-year-old man. The papules are arranged in a circular pattern. The lingual papillae appear normal. Skin lesions are also present.

Annular pattern: Circles and rings may be formed by the papular distribution. The lingual papillae in the area tend to undergo atrophic change, so that the centers of these rings are reddened and smooth (Fig. 6). The association of lichen planus annularis and smooth atrophy of the tongue was observed by Sayer.[30]

Verrucous pattern: Within a large area of involvement, there may be a hyperplastic mass of tissue with a verrucous or papillomatous appearance. This type of lesion is not common, and we have seen it developing only within areas of extensive involvement (Fig. 5).

Erosive or ulcerative pattern: This may be the result of vesicles rupturing and leaving a raw ulcerated area, but in rare cases these ulcerative lesions form without previous vesicles or bullae. They may represent secondary bacterial infection which causes epithelial destruction, but we are not quite sure of this. Biopsies and bacterial cultures in these areas are nonspecific. Adjacent to the ulcerated area, one can usually discern the white lines and papules of the typical lesions (Fig. 7). Ulcerative lesions have been described by many authors. Noguer and More[28] reported a case in which lingual ulceration developed slowly six months after the formation of typical white plaques.

Atrophic pattern: One of the more common lesions on the tongue involves the entire dorsum with the development of an atrophic glossitis. There is atrophy

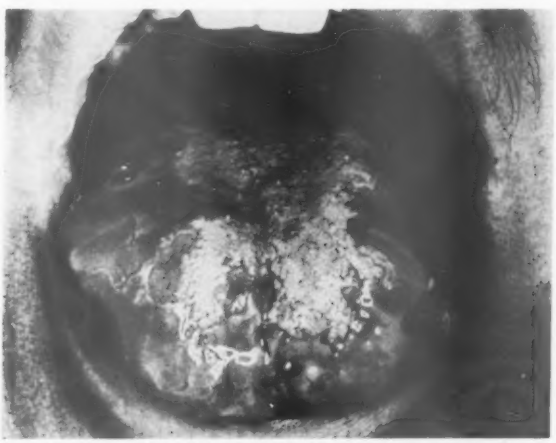

Fig. 7.—Lingual lesions, erosive pattern, in a 35-year-old man. Note the multiple smooth, ulcerated areas. No initial vesiculation was noted.

of the filiform and fungiform papillae, and there may or may not be white plaquelike lesions. In appearance, this form of lingual lichen planus is extremely similar to the atrophic glossitis of tertiary syphilis[14, 23] (Fig. 8). In the early literature these two lesions were often confused and, because of this, a relationship between lichen planus and syphilis was postulated. This relationship, in fact, does not exist, but these lesions are very similar in appearance

and only biopsy and serologic studies offer a definitive diagnosis in most cases. In atrophic lichen planus, since the lingual papillae disappear, the tongue appears reddened and often somewhat edematous. Atrophic lichen planus frequently tends to occur in elderly women. We have seen this in several cases. In one case two sisters presented atrophic lingual lesions of lichen planus, corroborated by biopsy.

Bullous or vesicular pattern: This is a rare variation of oral lichen planus, but in our experience its occurrence is more common on the tongue than in other areas. The lesions usually form on the lateral borders or undersurface of the tongue. The vesicles may vary in diameter from 1 to 2 mm. up to several centimeters. The larger vesicles or bullae are fluctuant and appear white or gray-purple in color. When the vesicle ruptures, its covering is seen to be very thick, due to the fact that the entire epithelium is lifted up. The fluid in the vesicles is usually clear, but this may be bloody or even purulent when the lesions become secondarily infected by oral microorganisms. Diagnosis of lichen planus from the vesicular lesions alone is difficult, and such vesicular eruptions as pemphigus vulgaris and benign mucous membrane pemphigoid, must be considered. However, more characteristic lesions of lichen planus are usually

A. *B.*

Fig. 8.—*A*, Glossitis of tertiary syphilis in a 64-year-old man. Note the smooth, glassy area at left and the raised keratotic pattern on the dorsum.
B, Lichen planus of tongue in a 40-year-old woman. Note the extreme similarity in appearance to syphilitic glossitis in *A*. Both cases were confirmed by biopsy studies.

present at the margins of the bullae or in other areas of the mouth. Biopsy specimens of the bullae are not helpful with respect to definitive diagnosis; tissue should be taken at the margins of the vesicular lesions. Kaposi,[16] in 1892, described bullous lichen planus as "lichen ruber pemphigoides," suggesting the clinical similarity to pemphigus. Strauss,[32] in 1933, reviewed seventy-eight cases of vesicular and bullous lichen planus in the literature and reported nine cases involving the oral mucosa. He suggested that the occurrence of bullae in

lichen planus was probably dependent on an excessive amount of edema enlarging the pre-existing microscopic lacunae or spaces that are usually present in lichen planus.

Plaques: Plaques occasionally may be seen on the tongue. These plaques tend to be gray-white and do not appear raised, since they replace the lingual papillae which atrophy and the adjacent papillae are more prominent than the lichen planus plaque. Indeed, on the dorsum of the tongue the plaques may appear somewhat sunken. These plaques are usually fairly dense and may present a patchy distribution throughout the tongue surface.

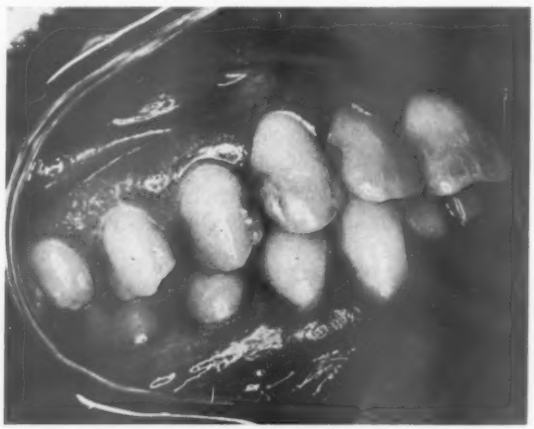

Fig. 9.—Gingival lesions of lichen planus, reticular pattern.

Lips.—Lichen planus seldom appears on the mucosal surface of the lips and, when it does, it usually is seen on the lower lip. These labial lesions are almost always accompanied by buccal lesions. In some cases the oral lesions may be confined to the lower lip. The discrete papular appearance is seldom seen on the labial mucosa.[27] The patterns usually seen are reticular, striated, and annular. Rarely, ulcerative or vesicular lesions may appear.

The lesions of lichen planus on the lips may simulate forms of cheilitis, and often the diagnosis is difficult in the absence of other lichen planus lesions. We have found also that labial lesions in the absence of other involved sites are difficult to diagnose clinically. Careful follow-up and biopsy are indicated.

Floor of the Mouth.—Lichen planus involvement is rare on the floor of the mouth, but cases have been reported in this exceptional location. We have seen a case in which there were reticular lesions of lichen planus on the floor of the mouth in a 13-year-old female patient.

Gingiva.—Lesions of lichen planus are occasionally found on the gingiva and have been described by numerous authors. We have found that gingival lesions occur on the attached portion of the gingiva and are usually of the

reticulated pattern. This involvement may extend onto the marginal gingiva in some cases (Fig. 9). In many cases there is a desquamative form of gingivitis associated with the gingival lesions of lichen planus. This interesting association has been reported previously.[7, 38] We feel that desquamative gingivitis is a nonspecific gingival manifestation of a variety of systemic disturbances, and it is not particularly surprising to find desquamative gingivitis associated with lichen planus. In the absence of other oral lesions, these gingival lesions could be confused with benign mucous membrane pemphigoid or other desquamating lesions of the gingiva. The reticular pattern often noted at the margin of the desquamating surface is helpful in rendering a diagnosis. Biopsy of the desquamative lesion usually reveals a nonspecific reaction. Enough of the attached gingiva should be included to show the reticulated area.

Skin.—The skin lesions of lichen planus are quite distinctive, and a clinical diagnosis is usually made without much difficulty. The primary lesion is a small, flat-topped, polygonal papule that varies from an erythematous appearance in the early stages to a more typical violaceous hue in the well-developed form. In the late stages a brownish color may predominate.

The most characteristic areas of involvement are the flexor surfaces of the extremities (Fig. 1), the sacral region, and the genitals. Trauma to the skin often is followed by the development of typical papules. This phenomenon may account for oral lesions at the interdental line on the buccal mucosa.

HISTOPATHOLOGY

The classic microscopic features of oral lichen planus lesions are parakeratosis, occasionally slight acanthosis, some downward extension of rete pegs, hydropic degeneration of the stratum germinativum, and an inflammatory infiltration of the connective tissue by a broad band of lymphocytes directly beneath the stratum germinativum of the epithelium. The deeper layers of the connective tissue are relatively free of chronic inflammatory cells, although dilated capillaries may be noted. The inflammatory infiltrate adjacent to the epithelium is composed primarily of lymphocytes, with scattered histiocytes and plasma cells often in evidence. The elongated rete pegs of the epithelium often appear in a "sawtooth" configuration; the picture is not common in mucosal lesions, however, although it is typical of skin lesions. In some cases there may be a hyperkeratosis instead of a parakeratosis, and in other cases a parakeratosis may appear adjacent to areas of hyperkeratosis. In both forms of keratinization there is an obvious accentuation of the stratum granulosum. Dyskeratosis is not a feature of lichen planus. We have not seen evidence of true dyskeratosis in any lesions studied.

The hydropic degeneration of the basal layer may involve a relatively small area, or it may involve the entire stratum germinativum. The degenerative changes characterized by liquefaction of the nucleus and often of the entire cell may extend to cells in the adjacent stratum spinosum. The protoplasmic intercellular bridges are often accentuated.

For microscopic diagnostic purposes, there should be the triad of (1) parakeratosis or hyperkeratosis, (2) hydropic degeneration of the stratum germinativum, and (3) chronic inflammatory infiltrate in the configuration of a broad band adjacent to the epithelium.

Lichen planus cannot be diagnosed on the basis of histopathology alone. There must be a correlation of both clinical and microscopic evidence. The clinical evidence includes the clinical appearance of the lesions, their distribution, and the course of the disease. The microscopic evaluation may then confirm the clinical impression. In writing up biopsy reports, we prefer to describe the lesion in microscopic terms and to state the diagnosis as "chronic inflammation consistent with a diagnosis of lichen planus." In the absence of clinical manifestations, it is doubtful that a case of lichen planus could be diagnosed even though the suspicion of its presence might be reasonably strong.

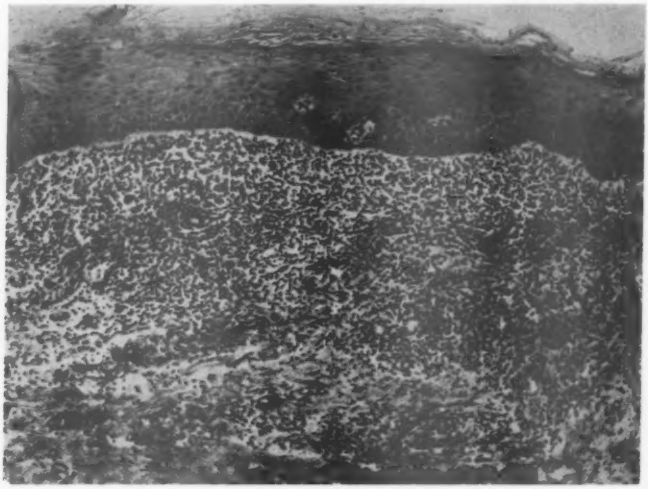

Fig. 10.—Low-power photomicrograph of biopsy specimen of white raised lesion from buccal mucosa. Note the parakeratosis, the regular stratum germinativum, and the even distribution of inflammatory cells in the underlying connective tissue. This initial biopsy revealed a nonspecific inflammatory reaction. (Hematoxylin and eosin stain. Magnification, ×70; reduced 3/10.)

It must be emphasized that the lesions of lichen planus often present a microscopic picture of nonspecific chronic inflammation. Lesions that at first show no evidence, microscopically, of lichen planus often develop later into a classic pattern. A case illustrative of this was seen by us in consultation. The lesions on the left buccal mucosa and retromolar pad suggested either lichen planus or leukoplakia to the patient's dentist. Biopsy revealed a somewhat thin epithelium with parakeratosis and a generalized infiltration of lymphocytes, plasma cells, and histiocytes into the connective tissue (Fig. 10). No evidence of hydropic degeneration was noted. The lesion returned after surgical removal, and biopsy studies at this time revealed the characteristic picture of lichen

planus (Figs. 11 and 12). In this case, as in similar cases, we believe that both lesions represented the oral manifestations of lichen planus but that the microscopic findings were at first nonspecific.

Fig. 11.

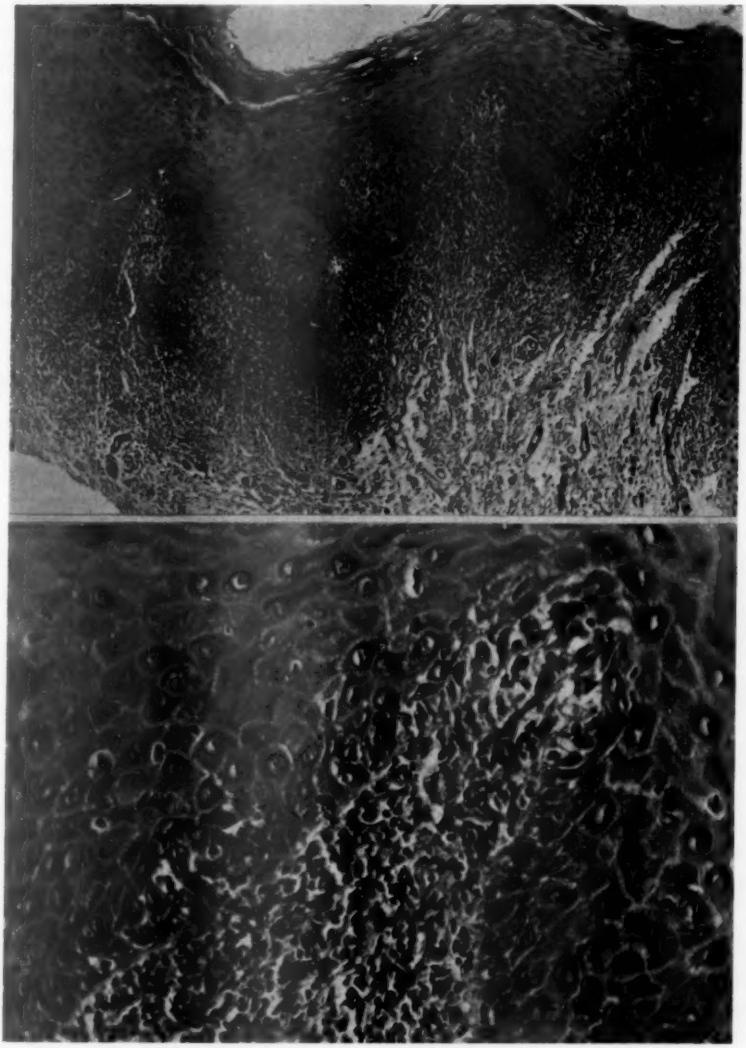

Fig. 12.

Fig. 11.—Low-power study of biopsy specimen from case represented in Fig. 10, taken three months later. Note the parakeratosis, irregularity of the stratum germinativum, and the dense band of inflammatory cells adjacent to the epithelium. Note also the relative absence of inflammatory cells in the deeper connective tissue. (Hematoxylin and eosin stain. Magnification, ×70; reduced 3/10.)

Fig. 12.—High-power study of section shown in Fig. 11. Note the hydropic degeneration of the stratum germinativum and the dense infiltrate of lymphocytes and histiocytes in the connective tissue. (Magnification, ×200; reduced 3/10.)

A negative microscopic report by no means rules out the possibility of lichen planus. Indeed, several cases with classic clinical manifestations presented a classic microscopic picture only late in the course of the disease or not at all.

We feel that biopsy studies are important, that they are necessary in cases presenting diagnostic problems, but that they are not essential in cases in which the clinical picture, distribution, and course of the disease are typical.

Recent studies in histochemistry have not added significantly to our knowledge of lichen planus. Steiner[31] has reported a distinct periodic acid-Schiff staining of the infiltrative reticulum in lichen planus, but he also pointed out that chronic inflammatory diseases of the skin tend to show some excess of mucopolysaccharides. Defects of basement membrane and increased periodic acid-Schiff staining of basement membrane have also been reported.[31]

In lichen planus bullosa the lesion has a subepithelial vesicle, as first pointed out by Poor.[29] Adjacent to the vesicular lesion there may be the classic microscopic features of lichen planus with the hydropic degeneration and subepithelial inflammatory infiltration. It is not difficult to follow the development of a subepithelial vesicle in lichen planus. The area of hydropic degeneration undergoes further disintegration and is replaced by a zone of fluid. The epithelium is lifted up as the area of fluid expands. The vesicle itself often contains scattered lymphocytes, polymorphonuclear leukocytes, and histiocytes.

In erosive lichen planus a vesicle has ruptured on the epithelium and has undergone necrosis. There is a typical picture of mucosal ulceration—a zone of fibrin densely infiltrated with polymorphonuclear leukocytes. Beneath this there is a zone of lymphocytes, plasma cells, and histiocytes, as well as fibroblastic activity.

DIAGNOSIS

The diagnosis of oral lichen planus can be made if several salient features are kept in mind. The primary lesion is a small papule, but these papules, in turn, may coalesce and align themselves to form several types of clinical variation. The disease is almost always asyptomatic. Only in the acute erosive or bullous variety are symptoms outstanding. Chronicity is the rule, and a course of several months to a year or more is typical. One should always inquire about the presence of skin lesions which may help to establish the diagnosis. Finally, a biopsy will usually disclose a characteristic histologic picture. The biopsy is very helpful, but it is not absolutely necessary in all cases. The clinical picture and history may be sufficient for a diagnosis in some cases.

Differential Diagnosis.—One should bear in mind certain other conditions that may occasionally resemble oral lichen planus very closely.

Chronic discoid lupus erythematosus often presents white, plaquelike, raised lesions with a border zone of erythema, and these lesions may be confined to the oral mucosa.[17, 22] The microscopic picture is often of considerable assistance in rendering a diagnosis. Microscopically, the lesions of discoid lupus erythematosus present a parakeratosis, some hydropic degeneration of the stratum germinativum, and some degeneration of the connective tissue collagen, characterized

by coagulation and increased staining. The connective tissue infiltration of chronic inflammatory cells tends to be perivascular in distribution, rather than subepithelial as in lichen planus.

Leukoplakia buccalis (leukokeratosis or smoker's patch) is one of the most common oral lesions and may resemble the plaque form of lichen planus. McCarthy[24] has divided leukoplakia into four grades according to clinical and histologic pattern. In the lower grades there are simple hyperkeratosis and chronic inflammation with a white plaquelike area that may be somewhat indurated. High-grade lesions present dyskeratosis or early malignant changes. Leukoplakia with evidence of dyskeratosis is to be considered a premalignant lesion. In the past lichen planus has been regarded as completely innocuous as far as possible development of carcinoma is concerned. In fact, many authors emphasize the importance of biopsy in lichen planus to rule out the potentially more serious leukoplakia. Lattes and associates[19] have recently reopened the question of oral lichen planus and cancer by reporting four cases in which cancer arose in erosive lesions of lichen planus. Those authors point out the rarity of this association but cast some doubt on the classically accepted benignity of lichen planus. Cases of carcinoma arising in mucosal lesions of lichen planus have also been reported by Carteaud and Stiegler[6] and others.

When the bullous or erosive form of lichen planus is present, the diagnosis may be quite difficult. *Benign mucous membrane pemphigoid* is perhaps the entity most similar in some aspects, especially in chronicity and lack of constitutional manifestations. However, lichen planus does not involve the ocular mucous membranes. At the periphery of the erosive areas there are definite infiltrative lesions in lichen planus which are absent in mucous membrane pemphigoid.

Pemphigus vulgaris in its early stages may resemble erosive lichen planus, but microscopic examination of the tissue would reveal the characteristic acantholysis with the formation of intraepithelial vesicles. Acantholysis is not seen in lichen planus, and the vesicles, when present, are invariably subepithelial.

Erythema multiforme may in certain cases resemble bullous lichen planus. Careful evaluation of the history and biopsy findings are of value in the differential diagnosis. The acute nature of erythema multiforme and the severe involvement of the labial mucosa are notable features.

Syphilis may also be considered in the differential diagnosis. The mucous patch of secondary syphilis may resemble a solitary plaque of lichen planus, but the former is usually quite friable and can be readily scraped away. Serologic tests must be ordered when the slightest doubt is present. The atrophic glossitis of tertiary syphilis may resemble the atrophic form of lingual lichen planus very closely. Biopsy usually reveals a sclerotic vascular picture in luetic glossitis, as well as perivascular lymphocytic infiltration.

Edema of the buccal mucosa may occasionally simulate hyperkeratotic lesions and is often produced by irritation from mastication. There is a grayish

discoloration, and a fine retiform pattern may be noted. This probably represents the attachment of the epithelium to the tunica propria. Application of pressure will obliterate the process by reducing the tissue fluid.

In recent years there has been mention of oral lesions of lichen sclerosus et atrophicus. An association of lichen sclerosus et atrophicus of the skin and lichen planus of the oral mucosa can occur, and we are inclined to doubt the existence of oral lichen sclerosus et atrophicus until clear-cut microscopic evidence is presented. An association of oral lichen planus and psoriasis of the skin may also exist. We have seen one case of this unusual association. Pigmentation following oral lesions is extremely rare, so that pigmented areas on the mucosa do not pose a diagnostic problem.

THERAPY

Treatment of lichen planus is at present far from satisfactory. The oral lesions of this dermatologic disease are more resistant to therapy than the skin lesions, and in cases of successful therapy the oral lesions are usually the last to disappear. Essentially, the therapy of lichen planus is empiric. Among various agents employed have been heavy metals, such as preparations containing bismuth, arsenic, mercury; vitamin B complex; antibiotics, such as penicillin; radiation; and immunization therapy. The corticosteroid hormones are being tried, but the results of this therapy are as yet indefinite. Supportive treatment, while important for skin lesions, is usually unnecessary in the management of oral lesions. As patients are nervous and depressed, we must attempt to regulate their existence so that diet is adequate and fatigue is prevented. Reassurance as to the nonserious nature of the disease is necessary.

In summary, therapy of oral lichen planus is unsatisfactory. Many therapeutic measures have been suggested over the years, but none are consistently effective. Usually no treatment is indicated. When discomfort is severe with the bullous form, corticosteroid therapy (both topical and systemic) will give symptomatic relief.

Since we feel that the emotional status of the patient is important, tranquilizers may be of some value. Reassurance is most important, as a high percentage of patients have cancerophobia. Since we are dealing with a self-limited disease, time is in our favor and patience will eventually be rewarded by a spontaneous cure.

REFERENCES

1. Anderson, N. P.: Lichen Planus With Vesicular Lesions on the Buccal Mucosa, Arch. Dermat. & Syph. **49**: 148, 1944.
2. Audry, M.: Sur un type clinique de lichen plan (lichen plan debutant sur les muqueuses), J. de Mal. Cut. et Syph. **8**: 12, 1894.
3. Barber, H. W.: Extensive Lichen Planus With Involvement of the Mucous Membrane in a Young Child, Proc. Roy. Soc. Med. **23**: 1929-30.
4. Bazemore, J., Johnson, H. H., Swanson, E. R., and Hayman, J. M.: Relation of Quinacrine Hydrochloride to Lichenoid Dermatitis (Atypical Lichen Planus), Arch. Dermat. & Syph. **54**: 308, 1946.
5. Bloom, D.: Lichen Planus of the Mucous Membranes in Two Sisters, Arch. Dermat. & Syph. **44**: 294, 1941.

6. Carteaud, A., and Stiegler, J. P.: Epithelioma sur lichen plan jugal, Bull. Soc. franç. de dermat. et syph. **57**: 320, 1950.
7. Cooke, B. E. D.: The Oral Manifestations of Lichen Planus: 50 Cases, Brit. Dent. J. **96**: 1, 1954.
8. Crocker, H. R.: On Affections of the Mucous Membranes in Lichen ruber vel planus, Monatschr. f. prakt. Dermat. **1**: 161, 1882.
9. Culver, G. D.: A Clinical Study of Lichen Planus, Arch. Dermat. & Syph. **1**: 43, 1920.
10. Dubreuilh, W.: Histologie du lichen plan des muqueuses, Ann. de dermat. **7**: 123, 1906.
11. Epstein, S.: Lichen Planus Confined to the Oral Cavity in Twins, Arch. Dermat. et syph. **45**: 328, 1942.
12. Fox, H.: Lichen Planus Confined to the Mouth, Arch. Dermat. & Syph. **32**: 150, 1935.
13. Gougerot, M. H.: Notions nouvelles sur le lichen plan des muqueuses buccolinguales, Rev. de stomatol. **38**: 856, 1936.
14. Gougerot, M. H., and Burnier: Lichen plan de la langue simulant la leucoplasie syphilitique, Bull. Soc. franç. de dermat. et syph. **36**: 1041, 1929.
15. Hallopeau, H., and Fouquet, J.: Lichen plan localisé a la levre inferieure, Bull. Soc. franç. de dermat. et syph. **13**: 250, 1902.
16. Kaposi, M.: Lichen ruber pemphigoides, Arch. f. Dermat. u. Syph. **24**: 340, 1892.
17. Lane, G. G.: A Case for Diagnosis—Lichen Planus? Lupus Erythematosus? Arch. Dermat. & Syph. **45**: 415, 1942.
18. Lebourg, M.: Lichen plan erosif de la face interne des joues, Rev. de stomatol. **41**: 556, 1939-40.
19. Lattes, Vrasse, Cernea, and Vigneuil: Lichen plan buccal et cancer, Rev. de stomatol. **54**: 58, 1953.
20. Lieberthal, D.: Lichen Planus of the Oral Mucosa, J. A. M. A. **48**: 559, 1907.
21. Little, E. G.: Lichen Planus, J. Cut. Dis. **37**: 639, 1919.
22. Little, E. G.: Lichen Planus? Lupus Erythematosus? Case for Diagnosis, Proc. Roy. Soc. Med. **23**: 1929-30.
23. Lortat-Jacob, L., Fernet, P., and Flandrin: Glossite atrophique par lichen plan aspect syphiloide, Bull. Soc. franç. de dermat. et syph. **36**: 1184, 1929.
24. McCarthy, F. P.: Etiology, Pathology and Treatment of Leukoplakia Buccalis, Arch. Dermat. & Syph. **34**: 612, 1936.
25. Milian, G., and Fouquet, J.: Lichen plan ulcere de la muqueuse jugale, Bull. Soc. franç. de dermat. et syph. **36**: 1218, 1929.
26. Montgomery, D. W.: A Remarkable Lichen Planus Lesion of the Tongue, Arch. Dermat. & Syph. **36**: 833, 1937.
27. Montgomery, D. W.: Lichen Planus of Lips, Arch. Dermat. & Syph. **38**: 401, 1938.
28. Noguer and More, M. S.: Lichen plan ulcereux de la langue, Bull. Soc. franç. de dermat. et syph. **44**: 665, 1937.
29. Poor, V.: Zur Anatomie der Schleimhaut Affectionen bei Lichen planus, Wilson, Dermat. Ztschr. **12**: 603, 1905.
30. Sayer, A.: Lichen Planus Annularis and Smooth Atrophy of the Tongue, Arch. Dermat. & Syph. **33**: 1174, 1937.
31. Steiner, K.: Mucoid Substances and Cutaneous Connective Tissue in Dermatoses. III. Cutaneous Mucopolysaccharides, J. Invest. Dermat. **28**: 419, 1957.
32. Strauss, M. J.: Vesicular and Bullous Lichen Planus, Acta dermat.-venereol. **14**: 447 1933.
33. Thibierge, G.: Lesions de la muqueuse buccale dans le lichen plan, Ann. de dermat. **2**: 65, 1885.
34. Trautmann, G.: Die Krankheiten der Mundhoehle und der oberen Luftwege bei Dermatosen, Weisbaden, 1911, J. F. Bergmann.
35. Unna, P. G.: Über die Mundaffektion bei Lichen ruber, Monatschr. f. prakt. Dermat. **1**: 257, 1882.
36. Wickham, L.: Sur un signe pathognomonique du lichen de Wilson, stries et ponctuations grisatres, Ann. de dermat. **3**: 517, 1895.
37. Wilson, E.: On Leichen Planus, J. Cut. Med. & Dis. Skin **3**: 117, 1869.
38. Ziskin, D. E., and Silvers, H. F.: Report of a Case of Desquamative Gingivitis and Lichen Planus, J. Periodont. **16**: 7, 1945.

ORAL ROENTGENOLOGY

American Academy of Oral Roentgenology

Arthur H. Wuehrmann, Editor

THE ROLE OF THE ROENTGENOGRAM IN THE DIAGNOSIS AND PROGNOSIS OF PERIODONTAL DISEASE

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INTRODUCTION

ORAL roentgenograms are considered essential for dental diagnosis, treatment planning, and prognosis, but their role in the study of periodontal disease is not generally understood. The diagnosis of periodontal disease is made with a periodontal probe, complemented by inspection and palpation. Although the dental roentgenogram plays a significant role in periodontal treatment planning and prognosis, the patient must be examined clinically before a diagnosis of periodontal disease can be made. This is true regardless of the roentgenographic findings.

The value of the roentgenographic examination is not disparaged by a critical appraisal of its limitations; the information that it furnishes is essential and can be obtained from no other source. If roentgenograms are to be used intelligently, their limitations should be known as well as their advantages.

LIMITATIONS OF ROENTGENOGRAMS

The limitations of dental roentgenograms in the diagnosis of periodontal disease are as follows:

1. Roentgenograms do not show the periodontal pocket.
2. They do not specifically distinguish between the successfully treated case and the untreated case.
3. They do not record the morphology of bone deformities.
4. They do not show the structures on the buccal or lingual aspects of the tooth.
5. They show no soft-to-hard tissue relationship.
6. They do not record tooth mobility.

Presented before the American Academy of Oral Roentgenology in Dallas, Texas, Nov. 9, 1958.

A periodontal pocket is a fissure between the cementum and the epithelium and is created by the destruction of periodontal attachment apparatus; it is roentgenolucent, and therefore it is not recorded on the roentgenogram. Pockets are found by probing the gingival sulcus around the entire circumference of the tooth (Figs. 1 and 2). The periodontal pocket is pathognomonic of periodontal disease; it is present in no other disease.

Fig. 1.

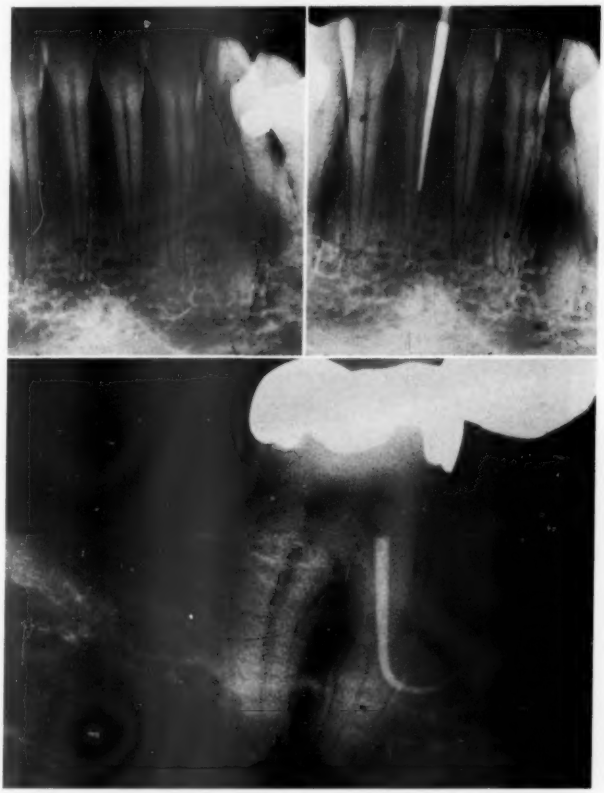


Fig. 2.

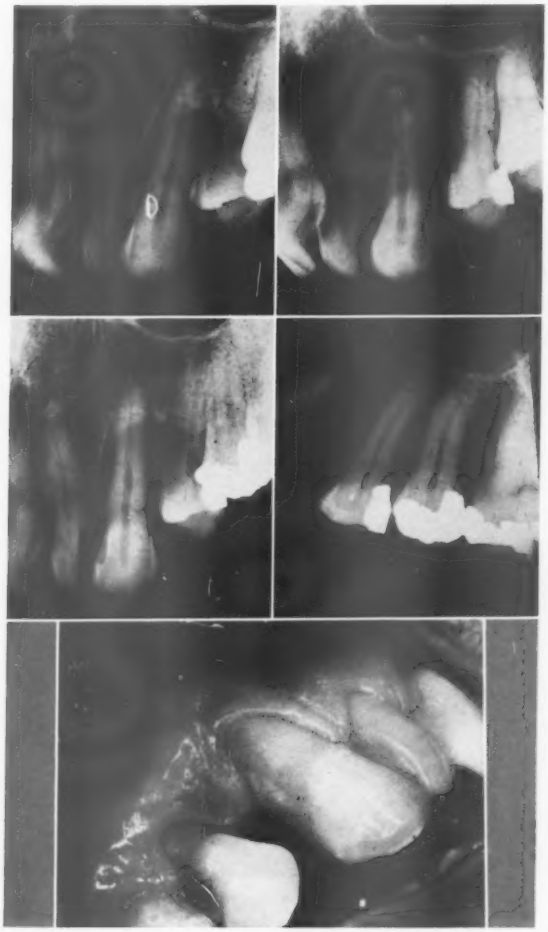
Fig. 1.—Roentgenograms of mandibular incisors with septal bone at normal level. Gutta-percha point reveals presence of deep pocket on labial aspect of right central incisor.

Fig. 2.—Roentgenogram of mandibular molar showing no evidence of periodontal disease. Gutta-percha point reveals pocket on buccal aspect of mesial root.

One of the results of periodontal disease, absorption of bone, may be seen on the roentgenogram in the septal regions, but this evidence is not proof that pockets are present (Fig. 3). Nor does the absence of bone absorption rule out the existence of pockets. Pockets may have been eliminated by therapy before the roentgenogram was made without changing the roentgenographic appearance of the alveolar process. Sometimes the gingivae recede

with the bone, and pockets do not form. Involvements of the furcation* are visible on the roentgenogram only after bone absorption extends beyond the furca. Then the shadow seen on the roentgenogram does not indicate whether

A.



B.

Fig. 3.—*A*, Roentgenograms of maxillary canine. The patient has never had periodontal therapy. *Above*, 1947; *below*, 1960. *B*, Photograph of region shown in roentgenogram. There is no pocket. The papilla is not attached, but a probe will not go beyond its base.

it is caused by an invasion of the interradicular space, with loss of septal bone, or by the destruction of marginal cortical plate covering the furca.[1]

Furcation* = The area or region lying between and at the base of two or more normal anatomically divided roots; *interfurca* = the area or region lying between and at the base of three or more normal anatomically divided roots of teeth; *interradicular space* = the space between the roots, from apex to base. (A. D. A. Bureau of Library and Indexing Service, J. Am. Dent. A. **41: 250-252, 1950.)

An extensive involvement may be interpreted as the result of absorption of both marginal cortical bone and septal bone in the interradicular space. The roentgenogram does not indicate whether the involvement is on the buccal aspect, the lingual aspect, or both. The location of the deformity can be determined with the periodontal probe.

A.

B.

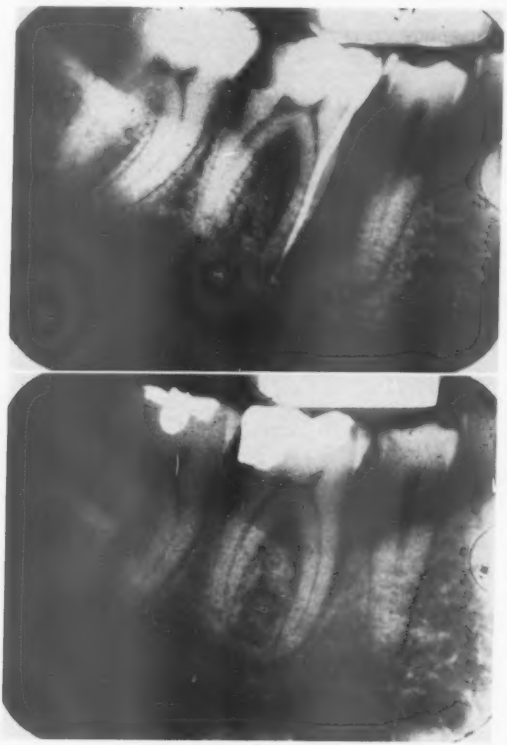
Fig. 4.—Roentgenograms of maxillary premolars before therapy to eliminate pockets *(A)* and eight years after therapy *(B)*. There are no pockets. The marginal bone is at different vertical levels on the buccal and lingual surfaces of the premolars. The roentgen rays pass through the inclined bony surface instead of across a crest.

The roentgenographic appearance of the alveolar process does not always show improvement after successful periodontal therapy, since lost marginal bone cannot be restored and an involvement of the furcation will not be filled with new bone (Fig. 4). The *intra*bony* deformity is an exception. Therapy can induce new bone to grow into such a space and eliminate the deformity, but the roentgenogram does not distinguish between an *intra*bony and an *infra*bony deformity (Figs. 5 and 6). Since the therapeutic requirements for

*The intrabony pocket is bounded by cementum on one side; opposite this is the epithelium lining the pocket, with epithelial attachment at the apical terminus of the pocket. An intrabony deformity is a defect in the bone surrounding the pocket.

osseous walls of deformities in the alveolar process are now well known,[2,3] the terminology should be specific. An *infra*bony pocket is a periodontal pocket in which the base of the pocket is apical to the crest of the bone; it may have one or more osseous walls.[1] This includes all of the osseous deformities that occur in periodontal disease. It would be better to describe the various deformities, such as craters and inconsistent margins, and to discontinue use of the nonspecific term, "infrabony."

A.



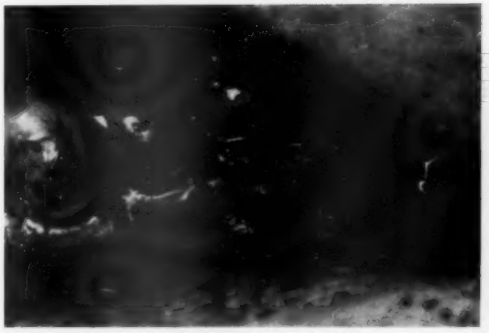
B.

Fig. 5.—Roentgenograms of an intrabony pocket before therapy *(A)* and after therapy *(B)*. The position of marginal bone on the buccal aspect is normal, but on the lingual aspect the margin is in a more apical position.

The term *intra*bony pocket means a pocket that is *inside* or *within* the bone. This term eliminates the necessity of saying "an infrabony pocket with three osseous walls" to identify a pocket that can usually be eliminated by creating a new growth of bone to fill the deformity. The roentgenogram will show definite evidence of osseous regeneration after successful *intra*bony pocket therapy, but osseous deformities that are apical to the crest but not *within* the bone can be eliminated only by ostectomy.[4-6]

Lost marginal bone will not be restored following periodontal therapy, but the diffuse roentgenolucent-appearing area of bone undergoing active absorption will be changed to a homogenous appearance after successful therapy. This change is due to the reduction of enlarged marrow spaces by the deposition of new bone. Friedman[7] has shown that this sometimes causes a false appearance of vertical bone growth, which explains what appears to be bone regeneration after treatment of bony deformities with one or two osseous walls. A crestal lamina dura may be seen on the roentgenogram after

A.



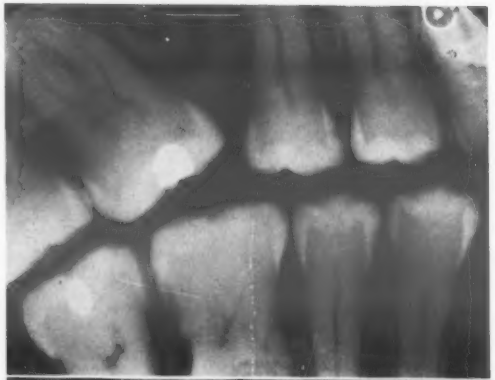
B.

Fig. 6.—*A*, Roentgenogram of bony defect does not reveal the number of osseous walls. *B*, Photograph shows that there is no osseous wall on the buccal aspect.

therapy, but this depends more on the topography of the bone and the angle of projection of the roentgen rays than on the degree of success achieved by therapy (Fig. 7).

The roentgenogram records vertical and horizontal planes, telescoping the width of the alveolar process with its topographic variations. It shows the vertical position but not the width of the alveolar process in the septal regions. The approximate depth of absorption may be seen, but neither the width nor the buccolingual position of the deformity can be recorded on the roentgenogram.

A.



B.

Fig. 7.

A.

B.

Fig. 8.

Fig. 7.—Roentgenograms made at the same appointment. *A*, "Bisect-the-angle" technique directs rays through the crest. The bony margins on the buccal and lingual surfaces are separated vertically. *B*, Roentgen rays directed across the crest superimpose bony margins and create a lamina dura on the film.

Fig. 8.—*A*, Photograph of interproximal bony crater. *B*, Roentgenogram does not reveal the crater.

The interproximal crater is the most common type of bone deformity occurring in periodontal disease; it is created by crestal absorption between the buccal and lingual cortical plates. If the marginal bone forming the walls of the crater is thin, the diffuse appearance of crestal bone undergoing absorption can be seen, since the walls will not be recorded on the roentgenogram. If the cortical plates forming the crater's walls are thick, their density will block out roentgenographic evidence of crestal bone absorption. In either case, the interproximal bony crater cannot be identified, for the roentgenogram shows a silhouette of bone and teeth in a single plane (Fig. 8).

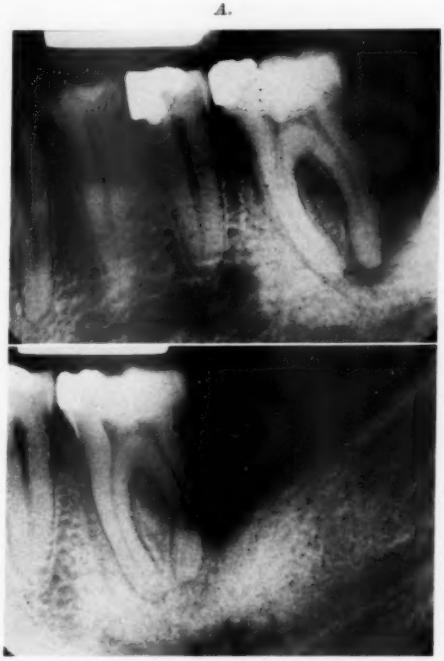


Fig. 9.—Roentgenograms of mandibular first molar with intrabony deformity extending beyond the apex of the distal root. The deformity was explored during therapy, and the defect beyond the apex was on the buccal aspect rather than directly under the apex. *A*, Before therapy; *B*, twelve months after therapy. (From Prichard: *D. Clin. North America*, March, 1960.)

Periodontal bone absorption which extends beyond the apex of the tooth usually involves the pulpal vessels in the pathosis, but the destruction can be lateral to the apical foramen. Therapy and prognosis will depend upon the exact position of the pathosis, and this cannot always be determined by studying the roentgenogram. Fig. 9 shows a lower molar with a shadow that appears to be under the apex of the distal root, but which exploration revealed to be on the buccal aspect of the root without invasion of the subapical region.

The tooth may be mobile in a buccal-lingual direction without showing roentgenographic evidence of mobility. The tooth root obliterates the image

of bone and related structures on the buccal and lingual surfaces where the greatest changes produced by periodontal traumatism usually occur.

BENEFITS OF ROENTGENOGRAMS

The benefits of roentgenograms in periodontal examination are as follows:

1. *With correct technique,* the position of marginal and septal bone on the tooth can usually be recorded.

Fig. 10.—*A,* Incorrect horizontal angle of projection causes area of bone absorption on maxillary second premolar to overlap the first premolar. Prognosis appears to be poor. *B,* Correct horizontal projection opens contact and reveals alveolar bone on the distal aspect of the first premolar; it has a favorable prognosis.

2. The roentgenogram acts as a monitor of the clinical examination; it may confirm a physical examination or suggest areas for such an examination, but it cannot, by itself, offer conclusive evidence.

3. The alveolar bone, the alveolar process, and the periodontal space on the mesial, distal, and apical aspects of the root are recorded on the roentgenogram in a single plane.

4. The clinical-crown-to-clinical-root ratio is documented.

5. Dense deposits of calculus and the margins of metallic restorations may be observed if they are on proximal surfaces of teeth.

To be of diagnostic value, the dental roentgenogram must show the tooth in its anatomic position in the alveolar process, so that the position of crestal bone can be determined. This can be accomplished only with the parallel or right-angle roentgenographic technique,[8] which includes accurate projection of the roentgen rays in both the vertical and horizontal planes (Figs. 10 and 11).

Fig. 11.—Roentgenograms of maxillary molars. *A*, Bisect-the-angle technique; area of bone absorption appears as halo on lingual root of first molar. Notice that the shadow does not extend above the apex. Prognosis appears to be hopeless. *B*, Vertical angulation superimposes zygoma over roots. *C* and *D*, Roentgenograms made by parallel technique. The shadow on the lingual root (*A*) is caused by a U-shaped area of bone absorption on the palatal surface of the lingual root. The vertical angle of projection magnified the defect and distorted the image. Prognosis is favorable.

Shadows cast in this manner will correctly record the vertical position of the teeth in the alveolar process; the root of each tooth will be recorded with a minimum of image foreshortening or elongation.[9-11] The roentgenogram will show the tips of molar cusps with little or none of the occlusal surface showing. If the roentgenogram shows the occlusal surface, the position of the marginal bone in relation to the tooth is not recorded correctely. This does not apply to mandibular premolars, since their buccal and lingual cusps are of unequal

height. The position of marginal bone on the tooth, as recorded on the roentgenogram, can be changed by the angle of projection of the roentgen rays, but the distortion is obvious to the dental diagnostician.

Additional features observed in high-quality diagnostic dental roentgenograms are distinct enamel caps and pulp chambers, open interproximal spaces, and proximal contacts that do not overlap. Special projections for specific regions, such as the distal oblique projection for maxillary third molars, close the embrasures, but they record diagnostic information in other than crestal areas.[12] Placing the film in the mouth correctly in relation to the teeth is just as important as the angle of projection, and this is the most difficult part of the oral roentgenographic technique. Careful processing is the final step in the production of diagnostic roentgenograms.[13, 14]

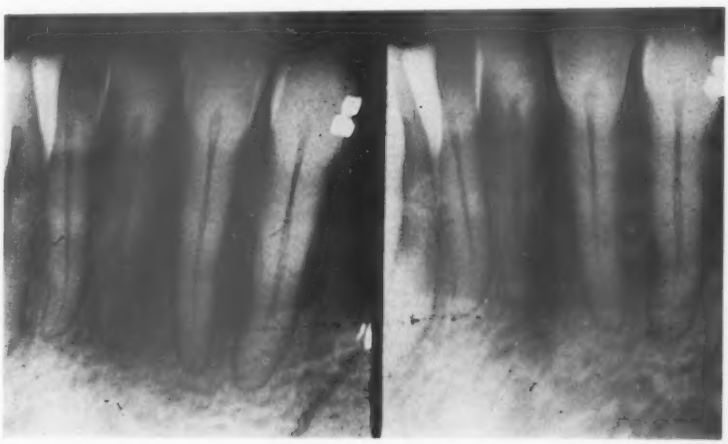


Fig. 12.—Roentgenograms showing evidence of bone absorption. Clinical examination revealed pressure from the lingual bar of a removable partial denture with destruction of gingiva and bone from the lingual aspect. Relief of pressure was the only therapy indicated.

Findings of the clinical examination must be incorporated into the roentgenographic interpretation; the latter acts as a check on the accuracy of the clinical procedures employed. The cause of roentgenographic evidence of bone absorption must be explained by the clinical examination,[15, 16] and through an understanding of basic biologic phenomena. Firm gingiva with good tissue tone and healthy surface characteristics may conceal the narrow aperture of a pocket which can be entered only with difficulty, and possibly with pain to the patient. Such pockets may be flask-shaped or tortuous. A tortuous pocket cannot be followed by a probe; it may have an insidious beginning on one root surface and become quite enlarged on another. The roentgenogram is a captious critic of the clinical examination; if bone and gingiva are not consistent with one another, the reason for inconsistency must be determined by the clinical examination (Fig. 12).

Periodontal traumatism causes changes in the alveolar bone* and periodontal space that may be visible on the roentgenogram. Evidence of periodontal traumatism may be seen as a widening of the periodontal space owing to absorption of alveolar bone (lamina dura), and as root absorption.[17] The alveolar bone and periodontal space on the mesial, distal, and apical aspects of the tooth are recorded on the roentgenogram.[18] The roentgenographic image of alveolar bone depends on the shape of the alveolus, and this is determined by the morphology of the tooth root. The lamina dura is indistinct around the maxillary incisors, since the roots of these teeth are round in cross section and the roentgen rays pass through the alveolar bone at a tangent.

Fig. 13.—Roentgenogram showing evidence of periodontal traumatism.

Mandibular incisors have elliptical or oblong-shaped roots in cross section, and the roentgen rays pass through the alveolar bone parallel with the greater dimension of the ellipse. Under normal conditions, this creates a distinct lamina dura on roentgenograms of these teeth. Fig. 13 shows a maxillary second premolar that is being subjected to trauma in a mesiodistal direction through a cantilevered pontic. This results in roentgenographic evidence of trauma that is not usually seen, because proximal contacts normally brace or splint teeth and thus limit movement in a mesiodistal direction. The width of the periodontal space varies with the amount of root movement. The hourglass appearance of the premolar socket in Fig. 13 is due to tooth movement at the apex and the neck of the tooth and to minimum movement at the center of tooth rotation. When teeth are moved bodily during orthodontic therapy, the periodontal space is uniformly enlarged.

The clinical-crown-to-clinical-root ratio is important in planning restorative treatment and in determining prognosis for individual teeth. This ratio is determined on the roentgenogram by comparing the portion of the tooth

*_Alveolar bone:_ The portion of the alveolar process which surrounds the alveoli in maxilla and mandible. It is a thin plate pierced by many small openings which transmit blood and lymph vessels and nerves. (_Dorland's Illustrated Medical Dictionary_, ed. 23, Philadelphia, 1957, W. B. Saunders Company.)

that is outside bone with the length and contour of the root that is invested in bone. This is one of the criteria for determining whether or not splinting is indicated.[19]

The roentgenogram may show evidence of overextension or underextension of restorations on proximal surfaces, and dense deposits of calculus on these surfaces may be recorded on the film. However, the roentgenogram cannot be used satisfactorily for the detection of subgingival calculus deposits.

Correctly projected and processed roentgenograms are *essential* for planning treatment and as aids in determining the prognosis of periodontal disease. Prognosis for the tooth involved in periodontal disease depends on the morphology of the bony deformity, surgical access to the deformity, the anatomy of the roots, and the functional demands that are to be placed upon the tooth. Roentgenographic findings are essential, but alone they are not definitive in any of these areas. The age and health of the patient are modifying factors.

The academic value of roentgenograms increases with their age; they should never be destroyed. When old roentgenograms are compared with recent ones, the rate of bone destruction by periodontal disease can be studied. This is an important factor in prognosis. Double film packets should be used, and one film from each packet should be retained permanently in the patient's file.

Because of roentgenographic limitations, surgical exploration during therapy is often necessary to determine the exact morphology of bony deformities. A provisional prognosis of questionable teeth may be made before treatment, with re-evaluation during surgical exploration and again after healing.

Definite rules cannot be established for prognosis, for the practice of health science is not an exact science; it is an art based on a science. *Judgment can be developed only by study and experience.* Thoma[20] defines prognosis as "the art of foretelling the duration, course, result, and termination of a disease." He states: "It depends largely on the correctness and accuracy of the diagnosis, but it is based principally on experience gained in practice." Prognosis cannot be arbitrarily determined by the level of remaining bone; the topography of the surrounding bone, rather than the quantity, is a determining factor. Also, bone that appears roentgenographically to be extremely dense may be less resistant to periodontal breakdown than less dense-appearing bone. The greater organic content of the less densely calcified bone is probably more favorable for a normal rate of bone maintenance. This observation has also been made with regard to the stability of teeth serving as abutments for removable partial dentures.[21] Furthermore, not all pockets are amenable to surgical interference; regional anatomy frequently makes pocket elimination impossible.

With the foregoing limitations in mind, prognosis of specific periodontal involvements can be discussed. Mandibular first molars with bifurcation involvements usually have a favorable prognosis. Mandibular second molars with similar involvements usually have a less favorable prognosis, since the regional anatomy limits access for therapy and oral hygiene. The proximity of the

ascending ramus of the mandible and the position of the external oblique ridge and muscle attachments on the buccal aspect of the second molar often make it impossible, even with modern techniques, to obtain an adequate zone of attached gingiva and sufficient vestibular depth for a favorable long-range prognosis.

Maxillary first premolars sometimes have bifurcated roots. Periodontal bifurcation involvement of these teeth often has a poor prognosis because of the inaccessibility of the bifurcation to oral hygiene measures.

Maxillary first molars with trifurcation involvements have a favorable prognosis if bone is still intact in the interfurca. Involvements from the mesiolingual aspect or from the buccal aspect have a favorable prognosis, since they are accessible for surgical resculpturing that makes adequate oral hygiene possible. Trifurcation involvements from the distal aspect are generally hopeless because of inaccessibility for purposes of good oral hygiene. However, if the tooth is the terminal one in the arch and the patient practices scrupulous oral hygiene, the distal trifurcation involvement may have a favorable prognosis. Prognosis for a trifurcation involvement of the second molar is less favorable than for a similar involvement of the first molar because of the second molar's smaller root structure, restricted interradicular space, and more distal position in the arch.

Mobility cannot be determined from the roentgenogram alone, but a widened periodontal space, alveolar bone absorption, and root absorption all indicate periodontal traumatism which may cause abnormal mobility. Prognosis depends on the cause of the mobility and on the possibility of its correction. If periodontal pockets and bone absorption are present because of periodontitis, in addition to periodontal traumatism, the prognosis is also influenced by the morphology of the bony deformity. Fixed splinting of mobile teeth may change an unfavorable prognosis to a favorable one.

Teeth involved in *intra*bony pockets (that is, pockets within the bone) have a favorable prognosis. *One of the reasons for frequent failure of intrabony pocket therapy is that the therapist bases his diagnosis of bone morphology entirely on the roentgenogram.* The bony defect with one or two osseous walls cannot be distinguished roentgenographically from an intrabony deformity. The vertical extent of bone absorption is recorded, but the width, position, and number of osseous walls cannot be seen on the roentgenogram. The tooth that is involved in an acute periodontal abscess usually has a more favorable prognosis than it would have with the same amount of bone loss if the lesion were chronic.[22] After reduction of the acute symptoms, the pocket is treated as indicated by the morphology of the surrounding bone. The architecture of deformities resulting from acute periodontal abscesses is often intrabony.

In addition to the factors discussed for prognosis of the individual tooth, the long-range prognosis for the dentition affected by periodontal disease depends on adequate home care and on the patient's resistance to periodontal disease; the latter, obviously cannot be determined by a roentgenogram.

SUMMARY

The diagnosis of periodontal disease involves clinical observation and the use of a probe; the roentgenogram is essential for planning treatment and for determining prognosis. The periodontal pocket is pathognomonic of periodontal disease, but the pocket is roentgenolucent and is not recorded in the roentgenogram. Evidence of bone absorption caused by periodontal disease is recorded on the film, but neither the morphology nor the lateral position of the osseous defect can be determined, since the roentgenogram shows a silhouette of bone and teeth. To be of value in the study of periodontal disease, roentgenograms must be made by the right-angle or parallel technique. When made properly, they become captious critics of the clinical examination.

REFERENCES

1. Prichard, J.: A Technique for Treating Infrabony Pockets Based on Alveolar Process Morphology, D. Clin. North America, pp. 85-105, March, 1960.
2. Prichard, J.: The Infrabony Technique as a Predictable Procedure, J. Periodont. **28**: 202-216, 1957.
3. Prichard, J.: Regeneration of Bone Following Periodontal Therapy, ORAL SURG., ORAL MED. & ORAL PATH. **10**: 247-252, 1957.
4. Friedman, N.: Periodontal Osseous Surgery: Osteoplasty and Osteoectomy, J. Periodont. **26**: 257-269, 1955.
5. Ochsenbein, C.: Osseous Resection in Periodontal Surgery, J. Periodont. **29**: 15-26, 1958.
6. Schluger, S.: Osseous Resection—a Basic Principle in Periodontal Surgery, ORAL SURG., ORAL MED. & ORAL PATH. **2**: 316-325, 1949.
7. Friedman, N.: Reattachment and Roentgenograms, J. Periodont. **29**: 98, 1958.
8. McCormack, D. W.: A Technical Procedure for Furthering Advancement Toward Anatomical Accuracy, J. California D. A. **13**: 89, 1937.
9. Fitzgerald, G. M.: Dental Roentgenology. I. An Investigation in Adumbration, or the Factors That Control Geometric Unsharpness, J. Am. Dent. A. **34**: 1, 1947.
10. Grøn, P.: A Geometrical Evaluation of Image Size in Dental Radiography, J. D. Res. **39**: 289-301, 1960.
11. Theilade, J.: An Evaluation of the Reliability of Radiographs in the Measurement of Bone Loss in Periodontal Disease, J. Periodont. **31**: 143-153, 1960.
12. Fitzgerald, G. M.: Dental Roentgenography. III. The Roentgenographic Periapical Survey of the Upper Molar Region, J. Am. Dent. A. **38**: 293, 1949.
13. Wuehrmann, A. H.: The Long Cone Technique, Practical Dental Monographs, pp. 1-30, July, 1957.
14. Wuehrmann, A. H.: Roentgenographic Interpretation of Dental Caries, Practical Dental Monographs, pp. 3-46, September, 1959.
15. Ennis, L. M., and Berry, H. M., Jr.: Dental Roentgenology, ed. 5, Philadelphia, 1959, Lea & Febiger.
16. Stafne, E. C.: Oral Roentgenographic Diagnosis, Philadelphia, 1958, W. B. Saunders Company.
17. Ritchey, B., and Orban, B.: The Crests of the Interdental Septa, J. Periodont. **24**: 34, 1943.
18. Goldman, H. M., Millsap, J. S., and Brenman, H. S.: Origin of Registration of the Architectural Pattern, the Lamina Dura, and the Alveolar Crest in the Dental Radiograph, ORAL SURG., ORAL MED. & ORAL PATH. **10**: 749, 1957.
19. Prichard, J., Simon, P., and Lorimer, J. W.: Periodontal Prosthesis in Occlusal Trauma, J. Periodont. **29**: 131-136, 1958.
20. Thoma, K.: Oral and Dental Diagnosis, ed. 3, Philadelphia, 1949, W. B. Saunders Company.
21. Applegate, O. C.: Essentials of Removable Partial Denture Prosthesis, ed. 2, Philadelphia, 1959, W. B. Saunders Company.
22. Prichard, J.: Management of the Periodontal Abscess, ORAL SURG., ORAL MED. & ORAL PATH. **6**: 474-482, 1953.

3833 CAMP BOWIE BLVD.

ROENTGENO-QUESTIONS

The other day, while I was using a tube such as the one described by Dr. Lincoln R. Manson-Hing in the October, 1959, issue of Oral Surg., Oral Med. & Oral Path., the tube touched the patient's cheek while an exposure was being made. The patient complained of a slight tingling and described the sensation as a mild electric shock. I have no explanation for this phenomenon, but naturally have been refraining from again touching the tube to the patient's face. I would appreciate your comments.

Three authorities were consulted. All agree that if the effect is real, and not psychological, the source of the current would have to be from a voltaic cell, static or friction electricity, or current leaking from the head of the machine. The first possibility of a cell formed by two different metals is not likely. Frictional electricity is a possibility if the cone is insulated from the grounded machine. If this tube is fastened to a plastic cone which fastens to the machine, the rubbing of the tube could build up a charge. The last possibility, that of faulty grounding, is good only if the cone contacts the metal of the head and if the ground of the machine is, in fact, faulty.

The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

ROENTGENO-ODDITIES

Pressure Resorption

THE accompanying roentgenograms suggest resorption of the distal root of the mandibular second molar by the impacted third molar. The 27-year-old patient presented no subjective symptoms. The second molar was positive to vitality tests.

John G. Whinery
Amarillo, Texas.

Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

ORAL PATHOLOGY

General Section

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ABORTIVE ATTEMPT AT CLEFT LIP

Report of a Case

Burke Megahan, M.D., Wheeling, W. Va., and
S. David Freedman, D.D.S., Atlantic City, N. J.*

A RECENT birth at this hospital is of unusual interest because of an extremely rare congenital anomaly. A cursory review of the literature has revealed no reported cases of this particular type of cleft lip.

A brief discussion of the embryology of the structure will serve as a review and orientation. The existence of a normal face depends on the normal development of a large number of closely integrated structures, and it is not strange that this area is a relatively frequent site of malformations.[1] Approximately one in every 700 newborn infants shows some type of clefting, and the cleft is more frequently on the left side than on the right.

The frontonasal process normally gives rise to the major portion of the forehead, the bridge of the nose, and the philtrum of the upper lip. It grows down and lies medial to the orbits and to the ingrowing maxillary processes derived from the first branchial arches. To form the upper lip, the upper surface of the medial portion of the maxillary process fuses with the lower margin of the nasal pits and the medial margin fuses with the lateral margin of the most inferior part of the frontonasal process.[1]

An ectodermal invagination which appears during about the fifth week of embryonic life, separates the lips from the bony portions of the upper and lower jaws. This separation begins before the fusion of the maxillary and frontonasal process is completed, and therefore subsequent failure of fusion may involve the lip, the maxillary arch, or both. It may be unilateral or bilateral.[1] It may vary in extent from a slight, scarcely discernible notch in the upper lip extending to the vermilion border to a deep, yawning carvernous defect extending into the nostril and accompanied by a complete cleft of the hard and soft palate, causing the oral and nasal cavities to communicate directly.[2]

From the Departments of Obstetrics and Oral Surgery, Wheeling Hospital.
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CASE REPORT

On April 14, 1959, a 26-year-old white woman was admitted to the obstetrical service of this hospital. The physical examination was entirely within normal limits. Her blood type was A with Rh factors C+D+E–. Both the patient and her husband were of German and English descent, and there was no history in either family of a cleft in the facial structure. Upon careful questioning concerning her pregnancy, the patient stated that she

Fig. 1.—Extraoral view showing the vermilion notch and the incision-like line running from the notch to the right naris. (Photograph reversed in developing.)

Fig. 2.—Note continuity of the labial mucosa and alveolar ridge. White line is tongue blade used to retract lip.

had suffered no physical violence and no mental trauma other than a strong feeling of distress caused by the death of her grandfather at approximately the time of conception. The monthly pregnancy examinations had been entirely within normal limits and uneventful. The delivery was spontaneous and uninvolved.

Physical examination of the newborn baby was within normal limits, except for a fine suturelike line extending from the right naris to the vermilion border of the lip. A small

. ''-shaped notch on the vermilion border arose in this area to meet the ''suture line.'' This notch was no more than 3 mm. in height (Fig. 1). Examination of the rim of the lip revealed no notching in this area. Intraorally, the mucosa of the lip was continuous and no defect was present. The alveolar ridge, as well as the hard and soft palate, was intact. Palpation of the palate revealed a hard, bony surface with no break in continuity (Fig. 2).

SUMMARY AND CONCLUSIONS

An extremely rare type of congenital defect in the facial clefting process has been presented. A cursory review of the literature reveals no similar case. It has been postulated that this anomaly represents an abortive attempt at cleft lip.[3, 4]

The authors wish to express their personal thanks to Drs. C. D. Messerly, K. K. Kline, and W. D. Leslie for their help and interest in preparing this case report.

REFERENCES

1. Potter, E. L.: Pathology of the Fetus and the Newborn, Chicago, 1952, The Year Book Publishers, Inc., pp. 464-465.
2. Thorek, M.: The Face in Health and Disease, Philadelphia, 1946, F. A. Davis Company, p. 175.
3. Bernier, J. L.: Personal Communication.
4. Thoma, K. H.: Personal Communication.

COMMISSURAL LIP PITS

Frank G. Everett, M.S., D.M.D., M.D., and*
*William B. Wescott, D.M.D.,** Portland, Ore.*

CONGENITAL pits or fistulas in the vermilion border of the lips have been described as occurring bilaterally and, more commonly, in the lower lip. Extensive and thorough reviews of this rare anomaly have been made by Hilgenreiner[1] and by Watanabe and associates.[2]

This report deals with an entity that is possibly related to the labial pits described in the literature, *commissural lip pits.* Although commissural lip pits have been noted by Kerr and his colleagues,[3] they have not, we believe, been reported or described in detail in medical or dental journals. Since September, 1958, we have seen ten cases of lip pits occurring at the angles of the mouth; eight of these cases will be described in this article.

CASE REPORTS

CASE 1.—Patient L. B., a white man of 35 years, presented himself for periodontal consultation, and during the examination it was noted that he had a pit at either angle of the mouth (Fig. 1).

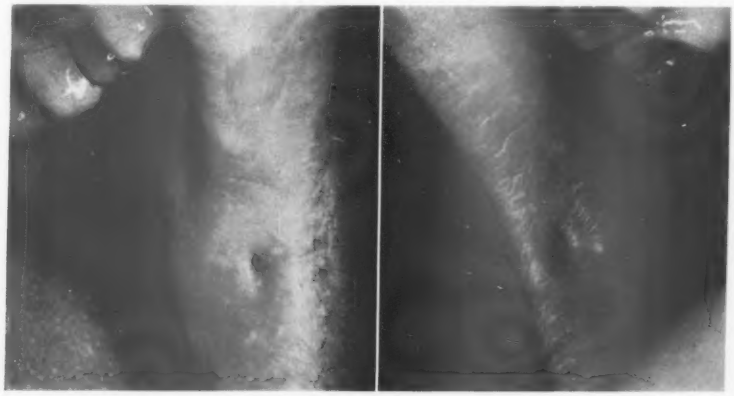


Fig. 1.—Case 1. Right and left commissural pits.

*Professor and Head of the Department of Periodontology, University of Oregon Dental School.
**Clinical Associate, Department of Periodontology, University of Oregon Dental School.

The fistulous openings were surrounded by a very slightly raised wall of tissue measuring about 2 mm. in diameter. The tracts diverged dorsolaterally, following the plane of the cheek.

The pits occasioned no discomfort at any time and posed no esthetic problem; in fact, the patient was not aware of their existence. The pits, measured with a periodontal probe, were 3 mm. deep (Fig. 2) on the left side and 4 mm. on the right side. Fluid could be expressed from the openings upon pressure; after drying, however, no additional liquid could be squeezed out. Physical examination and history revealed no other malformations. The patient refused to submit to a biopsy.

Fig. 2.—Case 1. Periodontal probe inserted into left pit. The depth indicated is 3 mm.

Fig. 3.—Case 3. Bilateral commissural pits.

CASE 2.—Mrs. A. S., a 47-year-old white woman and mother of Richard, Steven, Gilbert, and Karl S. (Cases 3, 4, 5, and 6), showed two commissural pits; the one on the right side measured 1.5 mm. and the one on the left measured 1 mm. No history of other anomalies or pertinent illness could be elicited in this patient or in any of her children.

CASE 3.—Richard S., 9 years old, had bilateral commissural lip pits. The right pit measured 3 mm. and the left one 4 mm. (Fig. 3). A biopsy specimen was taken, and the left pit or fistula was excised in toto for histologic examination.

Fig. 4.—Case 7. Commissural lip pit.

Fig. 5.—Case 8. Preauricular pit.

CASE 4.—Steven S., the 12-year-old son of Mrs. A. S., has what we should like to call commissural dimples.

CASE 5.—Gilbert S., the 25-year-old son of Mrs. A. S., also showed two commissural dimples.

CASE 6.—Karl S., the 23-year-old son of Mrs. A. S., showed two shallow bilateral commissural lip pits.

CASE 7.—Frances T., a white girl aged 11 years, presented herself for routine dental examination and was found to have commissural lip pits (Fig. 4). The pits were 2 mm. deep on the right and 1.5 mm. deep on the left side.

CASE 8.—Elaine T., the 12-year-old sister of Frances T., also was found to have bilaterally placed pits at the angles of the mouth. Their depths were measured as 1.5 mm. on the right side and 2 mm. on the left.

In both Cases 7 and 8 (Fig. 5) the patients also presented preauricular pits, bilateral in Case 8. The association of branchiogenic anomalies with other congenital malformations has been observed previously.[4]

Fig. 6 gives the genetic charts of families S. and T.

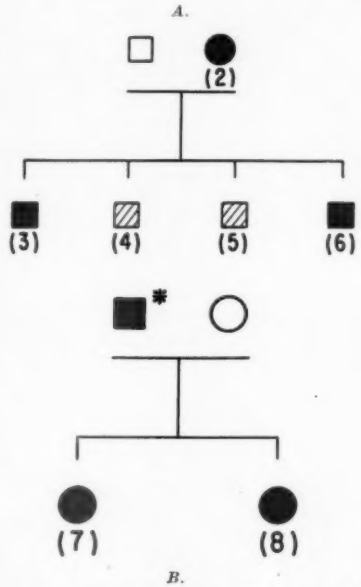


Fig. 6.—Genetic charts. *A*, Family S., Cases 2, 3, 4, 5, and 6. *B*, Family T., Cases 7 and 8. Case 7 shows also bilateral preauricular pits; Case 8 shows a preauricular pit on the right side only.

Square field = male; circle = female; solid field = commissural pits; cross-lined = commissural dimples; * = reported.

HISTOPATHOLOGY

In the sections of tissue studied (Case 3, Richard S.), it is evident that the course of the tract curved, resulting in a slightly tangential longitudinal section (Fig. 7, *A*) and a tangential cross section (Fig. 7, *B*). This indicates that the actual depth of the fistula was greater than that shown in our longitudinal section.

The low-power picture of the longitudinal section (Fig. 7, *A*) shows an invagination of the mucosa of the vermilion zone. The epithelium is many-layered, and pronounced hydropic degeneration and parakeratosis are evident

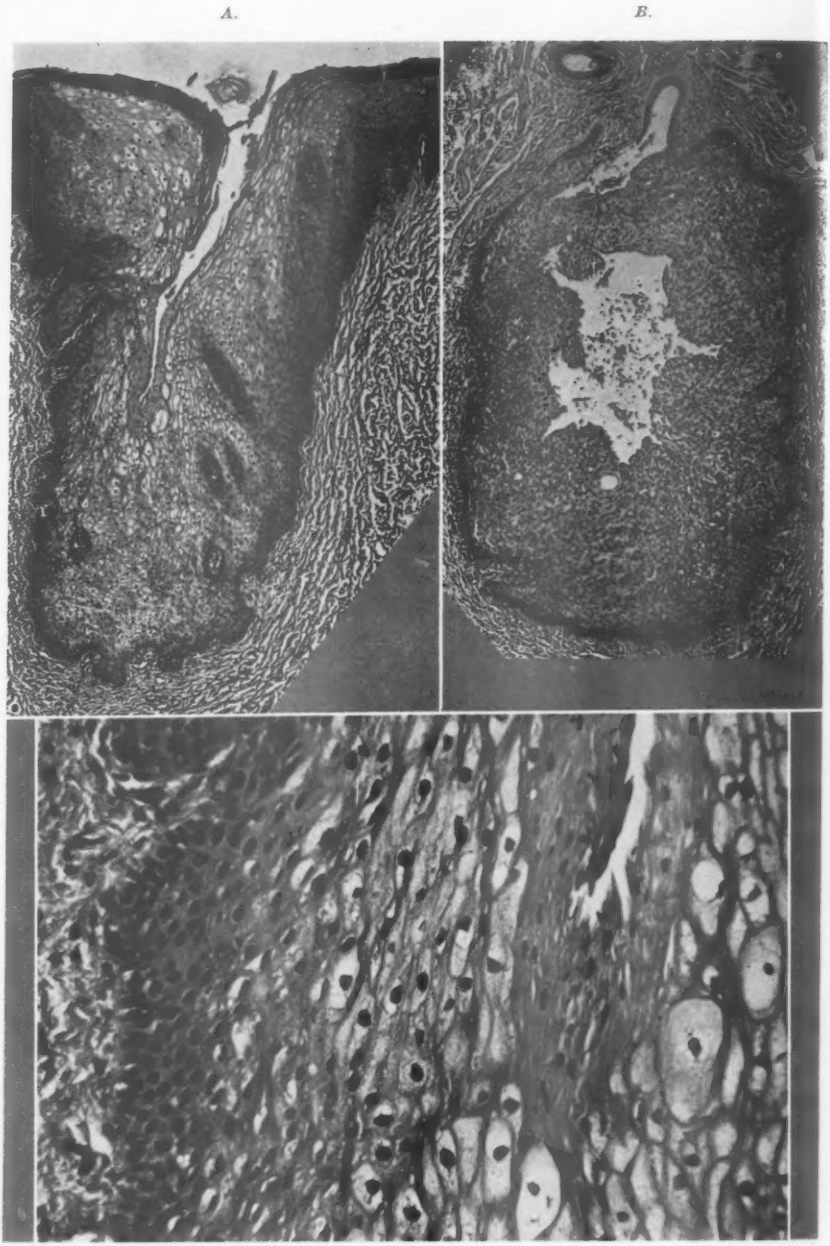
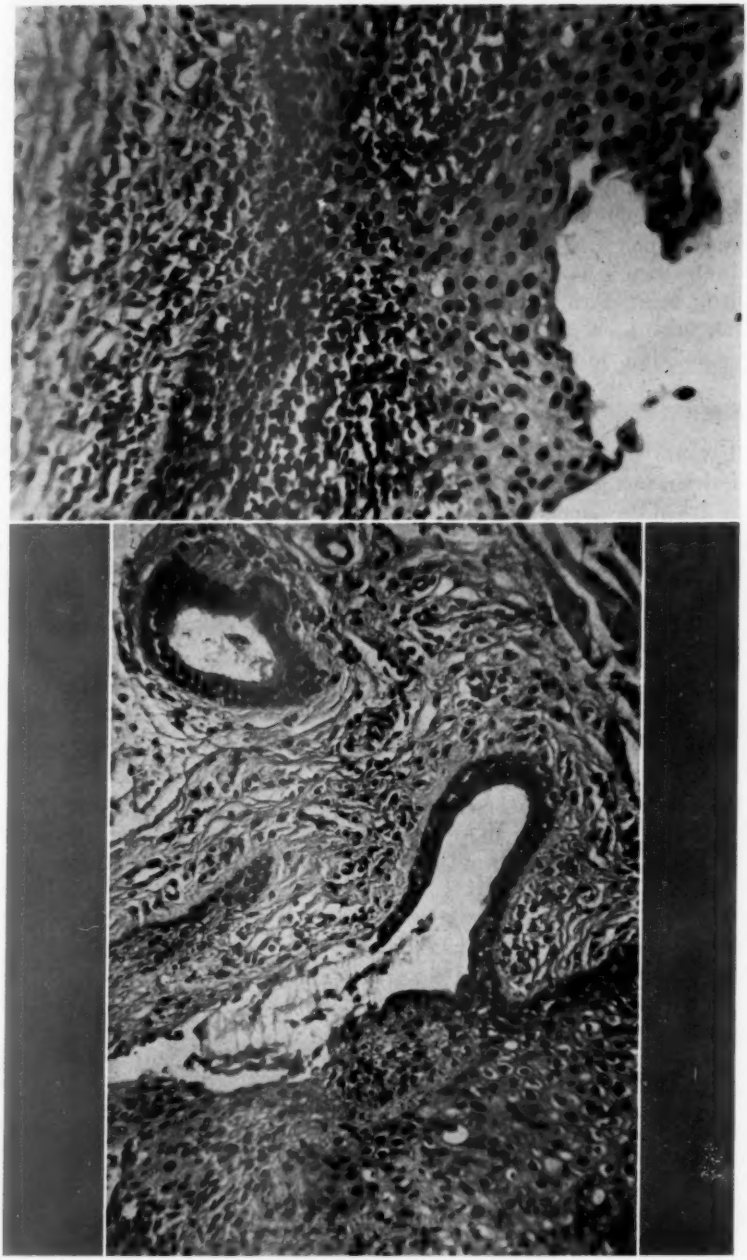


Fig. 7.—Case 1. *A*, Longitudinal section, slightly tangential. (Hematoxylin and eosin stain.) *B*, Cross section, slightly tangential. (Hematoxylin and eosin stain.) *C*, Higher magnification of section shown in *A*, showing parakeratosis and hydropic degeneration of the epithelium. (Magnification: *A*, ×85; *B*, ×100; *C*, ×450; each reduced 2/10.)

(Continued on opposite page.)

D.



E.

Fig. 7 (Cont'd).—*D*, Higher magnification of section shown in *B*. Pronounced subepithelial round-cell infiltration can be seen. *E*, Another field of the section shown in *B* at higher magnification showing the detail of the ductlike structure. (Magnification: *D*, ×400; *E*, ×300; each reduced 3/10.)

on higher magnification (Fig. 7, *C*). The cross section (Fig. 7, *D*) shows, on higher magnification, rather intensive subepithelial round-cell infiltration, particularly pronounced in a few spots.

In the deepest portion of the pit can be seen a structure, probably a duct (Fig. 7, *E*); when followed in serial section, it can be traced to its opening into the main lumen of the fistula. The duct consists of a double layer of cuboidal epithelial cells. There is a stringy, amorphous, eosinophilic material (probably mucin) in the lumen. Isolated leukocytes (salivary corpuscles) can also be seen in the lumen. Examining all our serial sections, we have been unable to connect this duct with any glandular tissue; our biopsy specimen may not have been large enough to include the appertaining gland structure. We assume, but do not know positively, that we deal with a true duct.

DISCUSSION

It would seem from the familial distribution of these cases that the mode of inheritance may be dominant, following the Mendelian laws.

In a random survey of 1,000 Portland school children during December, 1958, two cases of commissural lip pits were noticed. On this basis, it appears that the condition is not very rare but is overlooked, due perhaps to its location.

A study of the cases reported in the literature reveals a frequent association of congenital fistulas of the lower lip with other congenital defects.[5] It is interesting to note that the afflicted members of family T. also had preauricular pits.

None of the several theories that have been advanced to explain the development of lip pit anomalies have been generally accepted.[2, 6]

Commissural lip pits occur in a site similar to that of horizontal facial clefts, which are embryonal fissures separating the maxillary processes from the mandibular arch. When these structures approximate during development, their ectodermal coverings are brought into contact. The intervening ectoderm is then fenestrated and resorbed, and the processes become united by mesoderm, as suggested by Sicher[7] for fistulas of the lower lip. Perhaps it may be assumed that these commissural pits are formed when there "_____est une diminution de la force dynamique des masses mésodermiques séparées a l'origine par le mur épithélial."[8]

SUMMARY

1. Commissural lip pits, a condition that to our knowledge is as yet unreported in medical and dental journals, are described.

2. The anomalies are usually located symmetrically in the angles of the mouth.

3. The cases of eight patients with this abnormality are presented.

4. The condition may appear in several members of a family and in several generations, as shown in Cases 2 to 6, and Cases 7 and 8.

5. Preauricular pits were present in Cases 7 and 8; the other patients afflicted had no demonstrable congenital malformations.

The authors wish to thank Dr. Ellis B. Jump, Professor of Anatomy, University of Oregon Dental School, for the help they have received in the photomicrographic illustration of this paper.

REFERENCES

1. Hilgenreiner, H.: Die angeborenen Fisteln bezw. Schleimhauttaschen der Unterlippe, Deutsche Ztschr. f. Chir. **188**: 273-309, 1924.
2. Watanabe, Y., Igaku-Hakushi, M. O., and Tomida, K.: Congenital Fistulas of the Lower Lip, ORAL SURG., ORAL MED. & ORAL PATH. **4**: 709-722, 1951.
3. Kerr, D. A., Ash, M., Jr., and Millard, H. D.: Oral Diagnosis, St. Louis, 1959, The C. V. Mosby Company.
4. Jacobs, P. H., Shafer, J. C., and Higdon, R. S.: Congenital Branchiogenous Anomalies, J. A. M. A. **169**: 442-446, 1959.
5. Baxter, H.: Congenital Fistulas of the Lower Lip, Am. J. Orthodontics & Oral Surg. **25**: 1002-1007, 1939.
6. Gurney, C. E.: Congenital Pits of the Lower Lip, West. J. Surg. **48**: 753-754, 1940.
7. Sicher, H., and Pohl, L.: Zur Entwicklung des menschlichen Unterkiefers; ein Beitrag zur Entstehung der Unterlippenfisteln, Ztschr. f. Stomatol. **32**: 552-560, 1934.
8. Veau, V., and Politzer, G.: Embryologie du bec-de-lièvre; le palais primaire, Ann. d'anat. path. **13**: 275-326, 1936.

ENDODONTICS

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EFFECT OF VARIOUS PROCEDURES ON THE HUMAN DENTAL PULP

Pulp Reactions to Cavity Preparation and Gutta-Percha

Kaare Langeland, Ph.D., Oslo, Norway

INTRODUCTION

GUTTA-PERCHA has been used in operative dentistry partly for permanent fillings and partly for temporary fillings. Aware of the fact that teeth are usually rendered sensitive following insertion of gutta-percha in cavities in the dentine, some investigators have been concerned with the possible harmful effect of this filling material to the dental pulp. The main purpose of this study is to evaluate the reactions of the human dental pulp to gutta-percha and to observe the possibility of repair of the pulp tissues. Since cavity preparation is an inevitable procedure in any investigation concerning pulp reactions to filling materials, this factor will be taken into consideration as well.

PREVIOUS INVESTIGATIONS

Hellner,[1] among others, has examined the pulp reactions to gutta-percha in a histologic study. He found an inflammatory reaction in the pulp, characterized by hyperemia, stasis, edema, and the accumulation of leukocytes, plasma cells, and fibroblasts, as well as hyperproduction of capillaries. In dog experiments, Gurley and van Huysen[2] observed formation of secondary dentine and accumulation of round cells in the pulp tissue due to gutta-percha fillings. In similar experiments on human teeth, Rovelstad and St. John[3] found hyperemia, hemorrhages, round-cell infiltration, and possibly an increase in the rate of

From the Norwegian Institute of Dental Research, Josefinegate 32, Oslo, Norway.

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predentine formation. The observation concerning the predentine formation was in contradiction to the findings of Gurley and van Huysen,[2] who reported a reduction in the formation of predentine. Barker[4] reported results similar to those described by Rovelstad and St. John[3] after applying strong pressure to a worn burr and subsequently filling the cavity with gutta-percha. He concluded that drilling with a worn burr would not cause pathologic reactions in the pulp.

James, Schour, and Spence,[5] in an investigation concerning the response of the human dental pulp to cavity preparation and gutta-percha, observed a discontinuation of the pulp dentinal membrane and a reduction in the number of odontoblasts. These findings were not corroborated by the observations of Rovelstad and St. John,[3] who reported that there was no interruption in the regularity of the odontoblast layer. Furthermore, James and associates[5] observed an acute inflammatory reaction and, after increasing observation periods, a transition into a chronic stage. An invasion into the layer of Weil and the odontoblast layer by cells which those authors termed preodontoblasts was reported. The preodontoblasts were gradually converted into young and subsequently mature odontoblasts. A chronic inflammatory reaction persisted in the pulp, however, and repair was incomplete. As to the effects caused by cavity preparation, they concluded that the reactions would be similar, whether airbrasive or conventional burrs were used.

Massler[6] reported pulp damage under gutta-percha fillings due to the temperature rise that occurred during insertion of the filling and the margin leakage that took place almost immediately after the cooling.

STATEMENT OF PROBLEMS

It appears from the above-mentioned literature that some disagreement exists as to the interpretation of the histologic reaction caused by insertion of gutta-percha fillings in cavities in the dentine. One of the reasons seems to be that the investigators had not previously recorded the reaction to the cavity preparation per se, even if an attempt was made in one of the studies.[7] Accordingly, these authors observed a mixed reaction which was the result of cavity preparation, sterilization, and drying of the cavity as well as the gutta-percha itself. It was therefore considered important to record each step of this filling procedure, that is, the effect of the cavity preparation per se; the possible after effect of the cavity preparation; and the effect of drying the cavity. Finally, it was considered important to decide which reactions were really caused by the filling material in question. In case pathologic reactions were related to a specific cavity preparation method, it seemed desirable to avoid this procedure, and where the pathologic reactions could be traced back to the gutta-percha itself, it seemed of interest to decide what type of liner could prevent this harmful effect.

MATERIAL AND METHODS

These experimental series consist of seventy-five premolars of 9- to 16-year-old persons. Class V cavities were prepared in the gingival portion of the buccal

or lingual surfaces, which were all intact. In some cases Class I cavities were prepared. In some of the latter cases, occlusal fissure caries was observed before the start of the experiment.

The cavity preparation was performed with a conventional belt-driven engine (Ritter), an air turbine running at 50,000 r.p.m. (Dentalair), (Norlén),[8] or an air turbine running at about 250,000 r.p.m. (Ritter, S. S. White, Sterling, and Kavo-Borden air rotors). In cases where the purpose was to record the initial pulp reactions to a specific cavity preparation procedure, a continuous jet of water, a water-air spray, or a continuous jet of air was directed to the site of preparation, whereupon the tooth was immediately extracted and submitted to histologic investigation. In experiments in which the purpose was to record a possible after effect of the cavity preparation, zinc oxide (analytical reagent) and eugenol was subsequently inserted into the cavity and left there for varying observation periods before extraction and histologic examination.

In the series dealing with gutta-percha, a method that would not cause any initial reactions to the cavity preparations per se was used. The material subsequently inserted into the cavity was partly base plate gutta-percha and partly temporary stopping. The material was heated until plasticity was obtained, but it was not inserted into the cavity until it could be rolled between the fingers without burning. It was then pressed tightly against the cavity walls to assure as perfect a sealing as possible.

In some cases the bottom of the cavity was covered with a thin layer of gutta-percha, whereupon the rest of the cavity was filled with amalgam or zinc oxide-eugenol.

In other cases the bottom of the cavity was covered with a layer of zinc oxide-eugenol before insertion of gutta-percha.

In other cases liquid liners (Unifolan, Pulpdent, Copalite) were applied. Because chloropercha is known to adhere even to moist surfaces,[9] chloropercha also was tried as a liner.

In a last series gutta-percha was inserted into the experimental cavity and, after varying observation periods, replaced by another filling material (amalgam), according to a method by which the repair ability of the dental pulp can be studied.[10] In these last mentioned cases, cavities in two or more teeth of the same patient were filled with gutta-percha during the same sitting. After varying observation periods, one of the teeth was extracted and sectioned. The pulp conditions in the tooth or teeth left in the jaw would then be similar to those observed in the sections of the first extracted tooth, allowance being made for the effect caused by variations in cavity depth. The pulp conditions observed in the first extracted tooth would serve as a basis for evaluation of further development of the possible pulp reactions of the teeth left in the jaw.

When one tooth was extracted, the gutta-percha of the tooth or teeth simultaneously filled was replaced by a filling material assumed to be nonirritant—for example, zinc oxide (analytical reagent) and eugenol or amalgam. After varying observation periods, these teeth were extracted and submitted to histologic examination, and the pulp conditions were compared with those observed in the sections taken from the first extracted tooth.

HISTOLOGIC PREPARATION OF THE SPECIMENS AND CRITERIA FOR EVALUATION OF THE SECTIONS

The teeth were extracted under local anesthesia. After extraction, the root was cut off or holes were drilled through the root in order to facilitate penetration of the fixing agent. Decalcification in 5 per cent nitric acid was controlled by x-ray fluoroscopy and discontinued as soon as possible, whereupon the tooth was rinsed in water. The specimens were dehydrated in alcohol of increasing concentrations, from 70 up to 100 per cent. After two to five hours' clearing in benzol, the specimens were embedded in paraffin and cut in serial sections 4 to 5 microns thick, at least sixty to each tooth. The sections were dried in a thermostat, and alternate sections were stained with hematoxylin and eosin, iron hematoxylin, azan, or Masson's trichrome stain. They were embedded in Canada balsam, and the cover glass was applied under pressure.

In every case, each section was examined and the one revealing the shortest distance from the cavity bottom to the pulp, as measured along the dentinal tubules, was selected. The cavity-pulp distance was measured in these selected sections, while the general reaction was recorded in each section.

As for criteria for the histologic evaluation, it must be stressed that the observations made in sections taken from clinically intact untreated teeth of the same group as the experimental teeth cannot be unconditionally interpreted as indicating a pathologic reaction when observed in the experimental teeth. The possibility of artifacts must always be taken into consideration. A detailed evaluation of these phenomena has been published previously.[10]

OBSERVATIONS

Effect of Cavity Preparation Per Se and the Possible After Effect of Cavity Preparation.—In earlier experiments concerning pulp reactions to cavity preparation, it has been pointed out that the initial reaction, which with certainty could be recorded in the sections, was a displacement of the odontoblast nuclei, which in case of preparation under dry conditions appeared to have migrated into the pulpal end of the cut dentinal tubules.[10, 11] This reaction was avoided when a sufficient air-water spray or jet of water was directed to the cavity during cutting at 6,000 r.p.m. or at 50,000 r.p.m.[10]

In experiments in which air rotors running at about 250,000 r.p.m. were used, odontoblast nuclei appeared to have migrated into the dentinal tubules even if the air-water spray of these units was used at its maximal capacity.[11] In addition, erythrocytes as well were commonly observed in the dentinal tubules (Fig. 1).

When zinc oxide-eugenol had been inserted into the cavity immediately after preparation with the air turbine running at 250,000 r.p.m. and left there for some days, neutrophilic leukocytes appeared to have gathered along the predentine border in the area where odontoblast nuclei were seen in the dentinal tubules (Fig. 2).

In the area subjacent to the cut dentinal tubules, capillaries were dilated and filled with blood containing a considerable number of leukocytes (Fig. 2, *B*), whereas extravasated erythrocytes were scattered over the area (Fig. 2, *C*).

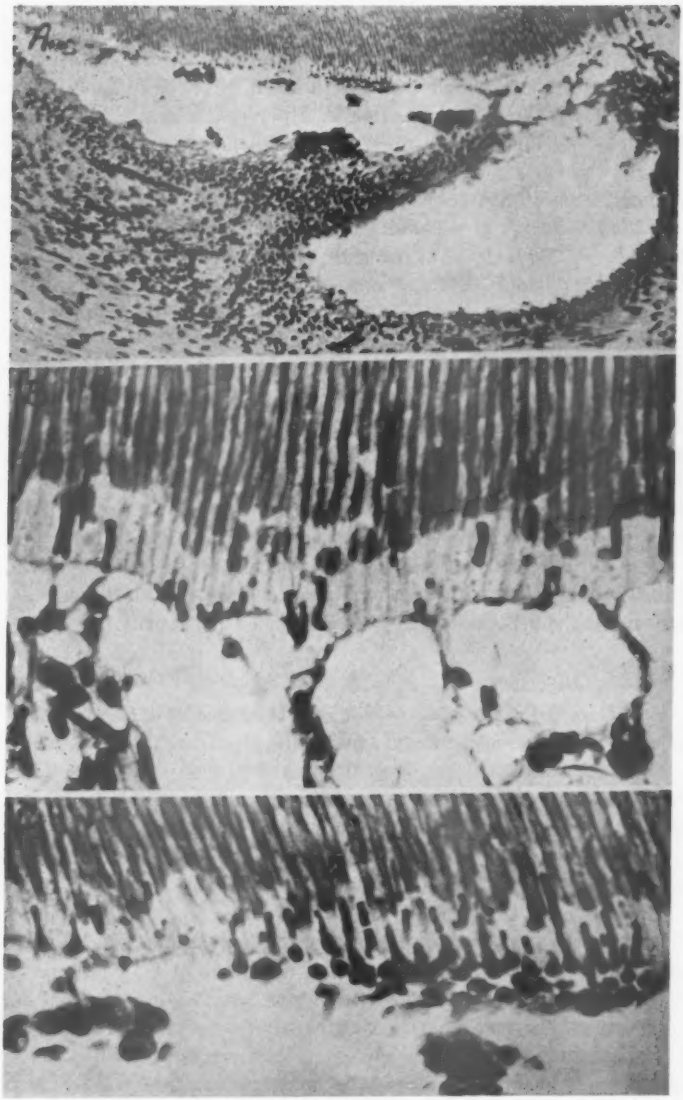

Fig. 1.—Cavity preparation with Kavo-Borden Airotor applying its maximum of air-water spray. Extraction immediately after cavity preparation. Cavity—pulp distance, 1.3 mm.

A, Area subjacent to cavity. Erythrocytes and odontoblast nuclei in dentinal tubules. Big cavity formation mostly an artifact.

B, Odontoblast nuclei in dentinal tubules.

C, Erythrocytes in dentinal tubules. Evidence of hemorrhage.

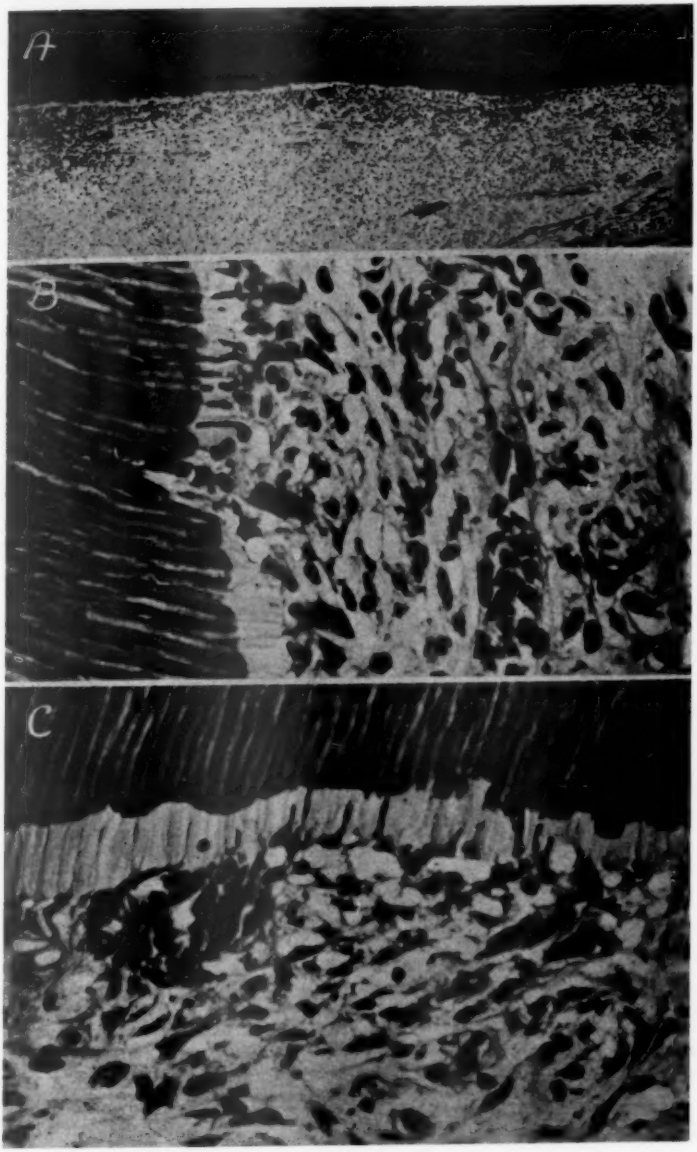

Fig. 2.—Cavity preparation with Kavo-Borden Airotor applying its maximum of air-water spray. Zinc oxide-eugenol. Observation period, fourteen days. Cavity—pulp distance, 1.3 mm.

A, Area subjacent to cavity. Accumulation of inflammatory cells in the odontoblast layer and subjacent pulp tissue.

B, Odontoblast nuclei in dentinal tubules, neutrophilic leukocytes in odontoblast layer and in vessels more centrally in pulp.

C, Ruptured capillary in odontoblast layer, extravasated erythrocytes (one in a dentinal tubule).

TABLE I. GUTTA-PERCHA

CASE NO.	AGE	TOOTH	DURATION (DAYS)	CAVITY-PULP DISTANCE (MM.)	ODONTOBLAST NUCLEI IN DENTINAL TUBULES	IRRITATION DENTINE	PRESENCE OF INFLAMMATORY CELLS		HYPER-EMIA	OCCURRENCE OF BROWN PIGMENT	FIG. NO.
							IN ODONTO-BLAST LAYER	CENTRALLY IN PULP			
1	10	+4	12 min.	1.6	Few	0	0	0	Slight	0	
2	11	5+	30 min.	2.8	0	0	0	0	0	0	
3	10	5−	1	1.3	Numerous	0	Numerous	Numerous	Strong	Frequent	
4	10	4+	1	1.2	Numerous	0	Numerous	Numerous	Strong	Frequent	
5	11	−4	1	1.6	Numerous	0	Some +	Numerous +	Strong	Frequent	
6	11	+4	1	1.6	Few +	0	Some	Some +	Moderate	Slight	
7	11	4−	2	1.3	Numerous	0	Numerous	Numerous	Strong	Frequent	4
8	15	5−	2	1.8	Some + and erythrocytes	0	Few	Some	Strong	Frequent	
9	12	+4	3	0.4	Some +	0	Few	Some +	Moderate	0	
10	11	+4	4	1.0	Numerous	0	Some +	Numerous	Strong	Slight	
11	11	4+	4	0.8	Numerous	0	Some +	Numerous	Strong	Frequent	
12	14	+4	5	2.6	Numerous and erythrocytes	0	Numerous	Numerous	Strong	Frequent	7, *A* and *B*
13	11	−4	5	1.9	Some +	0	Some +	Some +	Strong	Moderate	
14	12	+4	7	1.5	Some + and erythrocytes	0	Some +	Some +	Strong	Moderate	
15	12	4+	7	1.0	Few and erythrocytes	0	Few eosinophils	Some	Strong	Frequent	
16	14	+4	7	0.9	Some and erythrocytes	0	Numerous	Numerous	Strong	Moderate	
17	11	+5	7	1.5	Some +	0	Some +	Some +	Strong	Moderate	
18	13	+4	7	1.7	Numerous and erythrocytes	0	Numerous eosinophils	Numerous	Strong	Slight	
19	11	+4	14	1.6	Numerous and erythrocytes	0	Numerous eosinophils	Numerous	Strong	Frequent	5
20	11	+5	14	1.8	Few and erythrocytes	+	Some	Numerous	Strong	Slight	
21	11	4+	17	1.3	Few	+	Numerous	Some	Strong	0	

In cases in which zinc oxide-eugenol had been inserted into cavities prepared with the conventional engine applying an air-water spray, or with the air turbine running at 50,000 r.p.m. with its air-water spray, only slight insignificant reactions were recorded. These reactions are demonstrated in Fig. 3, A, in which case zinc oxide-eugenol had been placed in a cavity prepared with the air turbine running at 50,000 r.p.m. and using its maximum spray. The observation period was 304 days, and only a slight formation of irritation dentine revealed that a reaction had taken place.

A somewhat stronger reaction had taken place in cases in which cold air only had been applied during cavity preparation (Fig. 3, *B* and *C*).

Effect of Gutta-Percha.—It appears from Table I that all teeth filled with gutta-percha showed pathologic reactions in the pulp tissue. The histologic picture changed with increasing observation periods. After observation periods of a few days only, the most typical features of reaction were odontoblast nuclei in the pulpal ends of the cut dentinal tubules (Fig. 4, *A* and *B*), a break in the continuity of the pulp dentinal membrane (Fig. 4, *A*), and neutrophilic leukocytes along the predentine border (Fig. 4, *B*), in the odontoblast layer, and in the adjacent layer of Weil. The capillaries in the odontoblast layer were filled with blood, and in most cases extravasated erythrocytes were scattered over the area subjacent to the cavity (Fig. 4, *A*). Among the extravasated erythrocytes, neutrophilic leukocytes, lymphocytes, and a brown pigment were observed (Fig. 4, *D*) as well. The brown pigment appeared to be birefringent in polarized light (Fig. 4, *E*). In the vessels supplying the inflamed area, a considerable increase in leukocytes was observed (Fig. 4, *C*).

Circulatory disturbances were marked after fourteen days as well (Fig. 5). Dilated capillaries were seen in the odontoblast layer under the cavity (Fig. 5, *A* to *C*), deforming the odontoblast layer and the predentine (Fig. 5, *C*). In addition, the vessels supplying the inflamed area with blood seemed dilated and contained a considerable number of lymphocytes (Fig. 5, *D*). In the dentinal tubules only a few odontoblast nuclei were observed, and at the same time neutrophilic leukocytes were no longer conspicuous in the odontoblast layer, even if some were still present (Fig. 5, *C*). More centrally in the pulp, particularly along the vessels adjacent to the inflamed area, some eosinophilic leukocytes and increasing numbers of lymphocytes and transition forms of inflammatory cells were observed (Fig. 5, *D*).

The cases in which the cavities were lined with gutta-percha and sealed with amalgam (Table II, *A*) or zinc oxide-eugenol (Table II, *B*) revealed reactions similar to those described above (Fig. 6, *A* to *C*). On the other hand, no reactions occurred in those cases in which the cavities were lined with a thick layer of zinc oxide-eugenol (Table II, *C*). A comparison of Cases 22 and 28, two teeth from the same patient, demonstrates this phenomenon (Fig. 6, *A*, *B*, and *D*). If the layer of zinc oxide-eugenol was not sufficiently thick, however, a slight reaction occurred (Fig. 7, *C*, Case 30). On the other hand, this reaction was considerably reduced as compared with the one observed in the sections taken from another tooth (Case 12) of the same person, in which case gutta-percha was applied without a liner (Fig. 7, *A* and *B*).

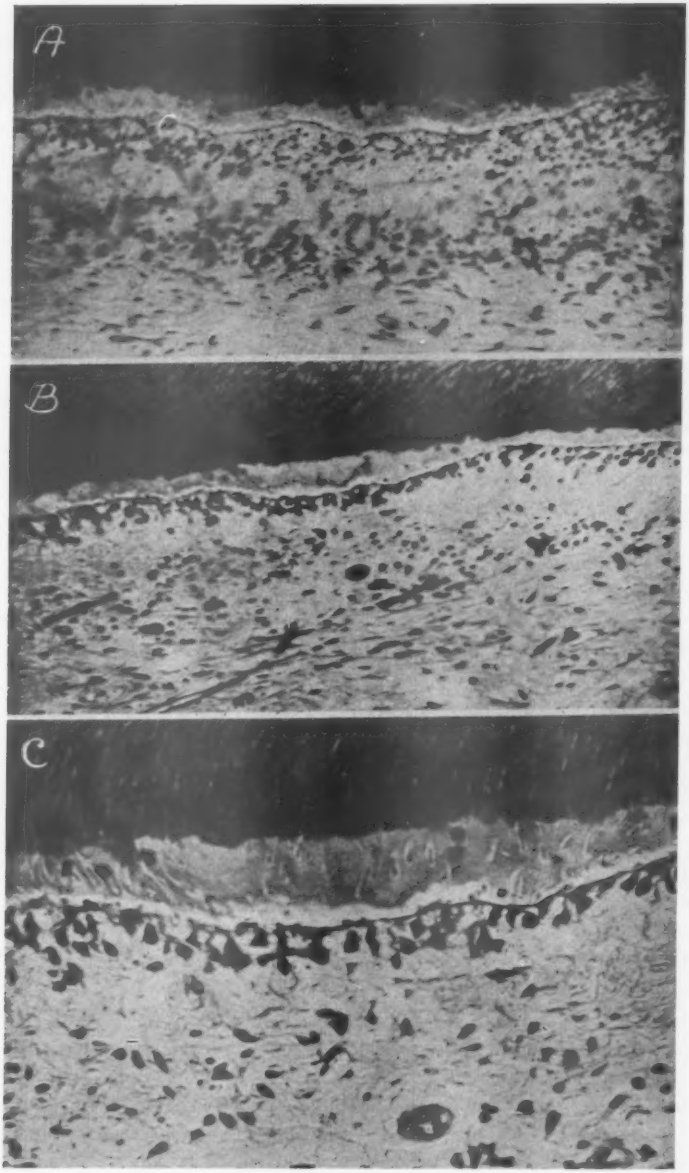

Fig. 3.—Cavity preparation with Dentalair applying its maximum of air-water spray. Zinc oxide-eugenol. Observation period, 304 days. Cavity—pulp distance, 0.9 mm.

A, Inconspicuous formation of irritation dentine in spite of deep cavity.

B, Same procedure as in *A*, but air-water spray has been replaced with cold air only. Observation period, 304 days. Cavity-pulp distance, 1.5 mm. Irritation dentine formed under cavity indicating initial deterioration of odontoblasts.

C, High magnification of same area. The irritation dentine irregularly formed, contains a reduced number of dentinal tubules corresponding to the reduced number of odontoblasts.

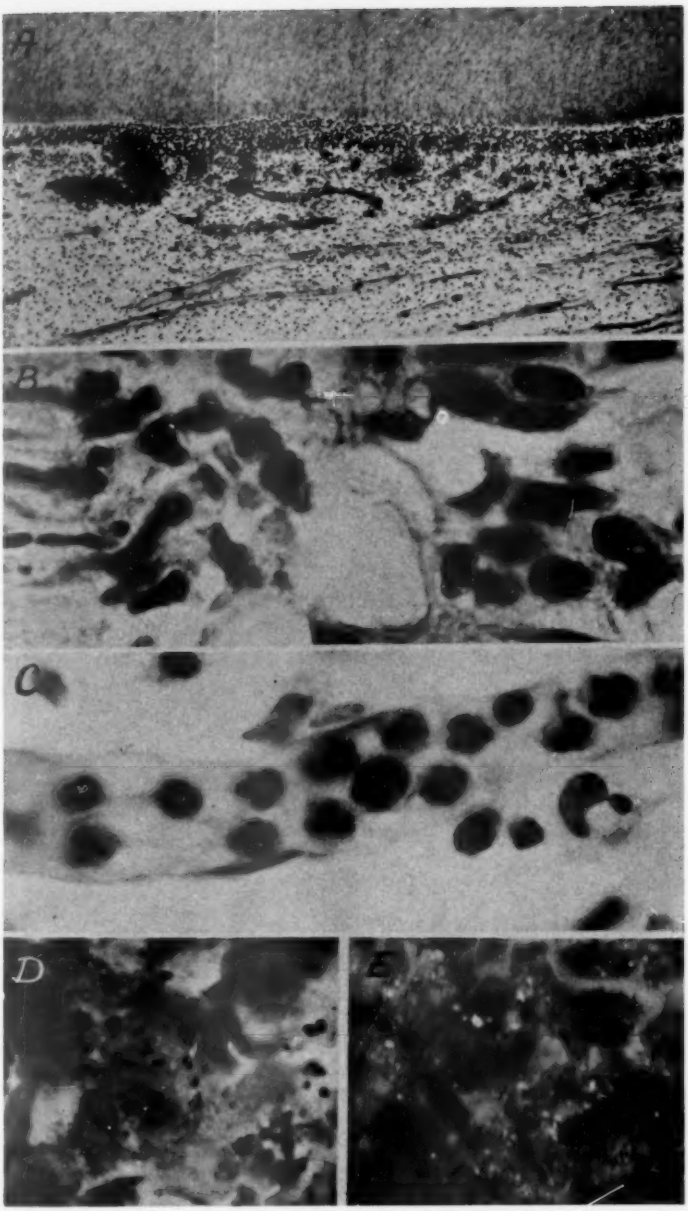

Fig. 4.—Case 7. Gutta-percha. Observation period, two days. Cavity-pulp distance, 1.3 mm.

A, Circulatory disturbances, accumulation of leukocytes. Numerous odontoblast nuclei in dentinal tubules.

B, Odontoblast nuclei in dentinal tubules. Neutrophilic leukocytes along predentine. Remaining odontoblasts to the right.

C, Vessel centrally in the pulp, containing mostly neutrophilic leukocytes and one eosinophilic leukocyte.

D, Brown pigment and extravasated erythrocytes.

E, Brown pigment birefringent in polarized light.

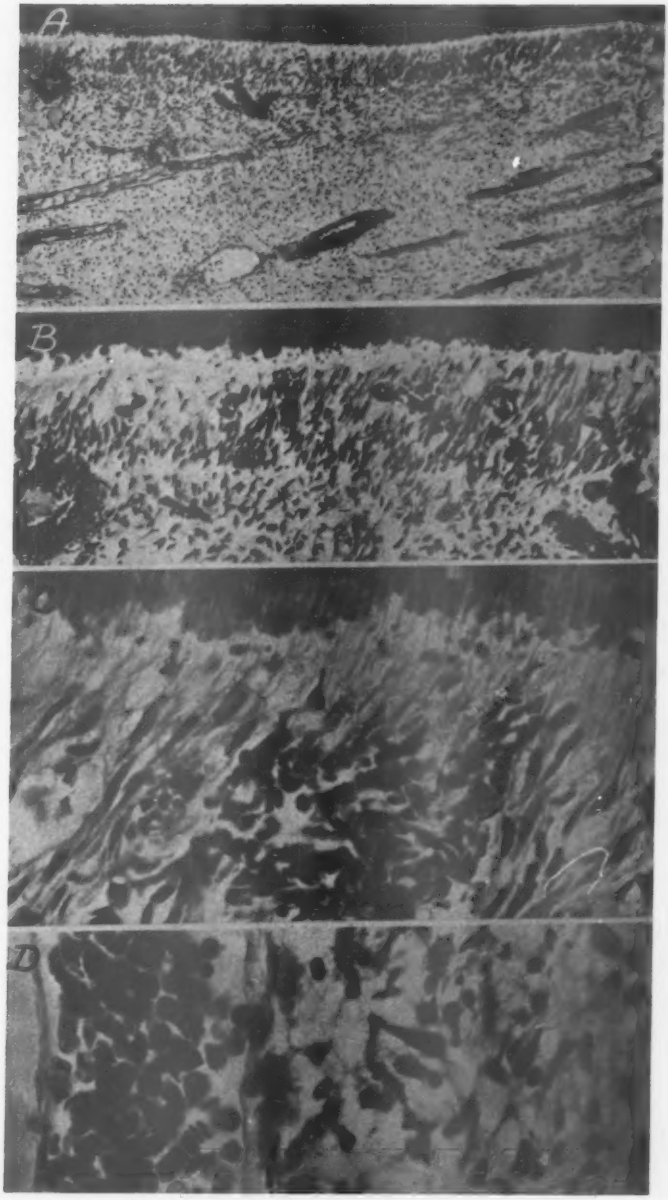

Fig. 5.—Case 20. Gutta-percha. Observation period, fourteen days. Cavity-pulp distance, 1.8 mm.

A, Circulatory disturbances, accumulation of inflammatory cells.

B, Dilated capillaries in odontoblast layer, congestion of blood in vessels more centrally in the pulp.

C, Ruptured vessel in odontoblast layer. Broad layer of irregular predentine.

D, Vessel centrally in pulp containing conspicuous number of leukocytes. Lymphocytes and transition forms of inflammatory cells in vicinity of vessel.

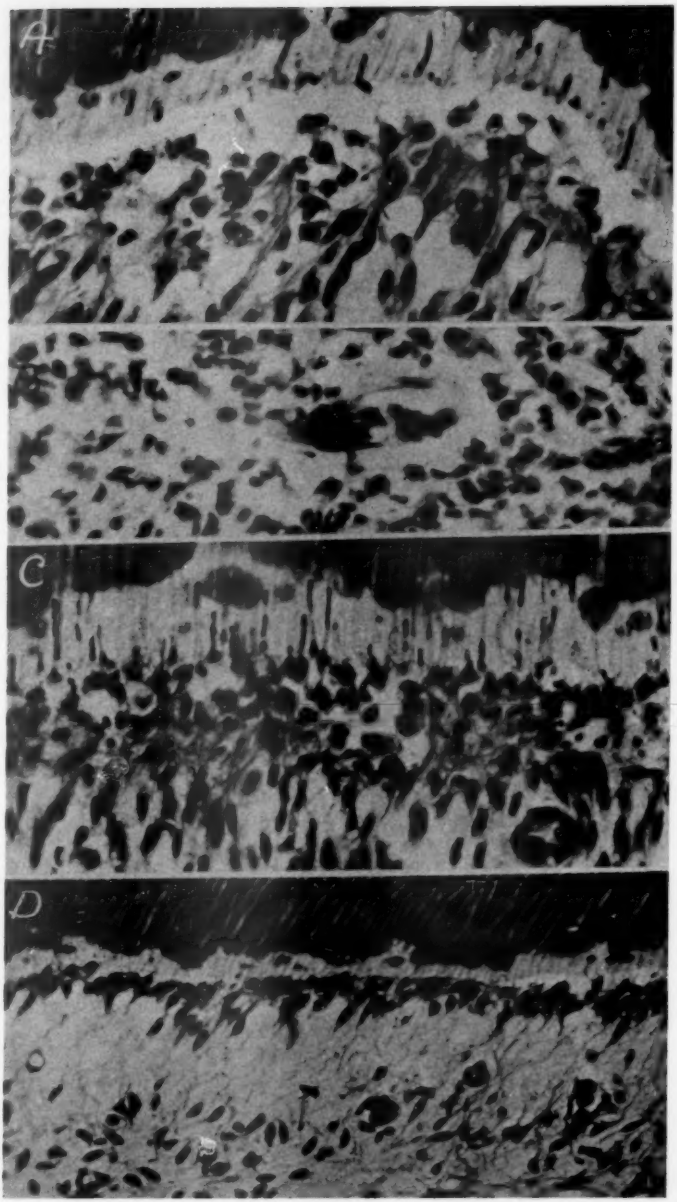

Fig. 6.—*A* and *B*, Case 22. Gutta-percha sealed with amalgam. Observation period, two days. Cavity-pulp distance, 1.4 mm. *A*, Odontoblast nuclei in dentinal tubules. Neutrophilic leukocytes in odontoblast layer. *B*, Extravasated erythrocytes and lymphocytes centrally in pulp.

C, Case 26. Gutta-percha sealed with zinc oxide-eugenol. Observation period, two days. Cavity-pulp distance, 1.7 mm. Odontoblast nuclei in dentinal tubules, abundance of neutrophilic leukocytes in odontoblast layer. Strong hyperemia.

D, Case 28. Zinc oxide-eugenol sealed with gutta-percha. Observation period, two days. Cavity-pulp distance, 0.7 mm. Normal conditions.

TABLE II

	CASE NO.	AGE	TOOTH	DURATION (DAYS)	CAVITY-PULP DISTANCE (MM.)	ODONTOBLAST NUCLEI IN DENTINAL TUBULES	IRRITATION DENTINE	PRESENCE OF INFLAMMATORY CELLS		HYPEREMIA	OCCURRENCE OF BROWN PIGMENT	FIG. NO.
								IN ODONTOBLAST LAYER	CENTRALLY IN PULP			
A. Gutta-percha sealed with amalgam	22	9	−4	2	1.4	Numerous	0	Numerous	Some	Moderate	Slight	6, *A* and *B*
	23	13	−4	2	1.2	Numerous	0	Some +	Some +	Strong	Moderate	
	24	13	+4	4	1.4	Some and erythrocytes	0	Some eosinophils	Some	Strong	Frequent	
	25	12	−4	5	1.5	Numerous and erythrocytes	0	Some	Numerous	Strong	Moderate	
B. Gutta-percha sealed with zinc oxide-eugenol	26	15	5+	2	1.7	Numerous	0	Numerous	Numerous	Strong	Slight	6, *C*
	27	15	5−	2	1.5	Numerous and erythrocytes	0	Numerous	Numerous	Strong	Slight	
C. Zinc oxide-eugenol sealed with gutta-percha	28	9	4+	2	0.7	0	0	0	0	0	0	6, *D*
	29	12	4+	2	0.8	Few	0	Tear O	Some	0	0	
	30	14	4+	5	2.1	Some	0	0	0	Slight	Slight	7, *C*
	31	10	4−	7	1.8	Few	0	0	Few	Moderate	Slight	
	32	10	+4	14	1.3	Some	0	Few	Some	Moderate	0	
	33	10	−4	42	1.8	0	0	0	0	Moderate	0	

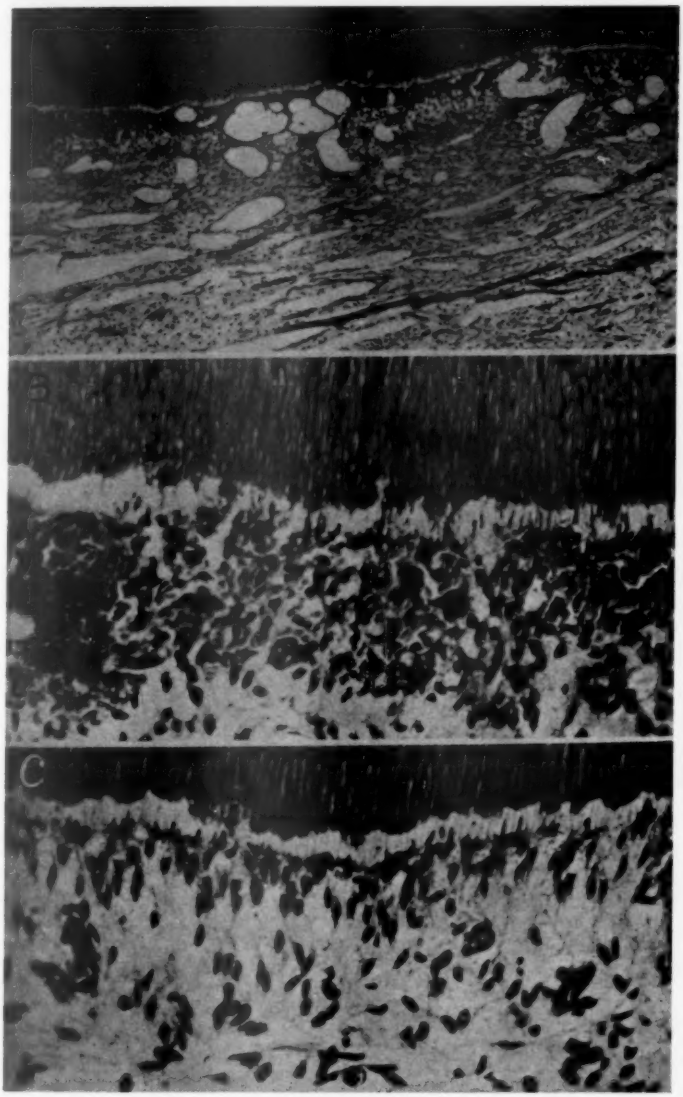

Fig. 7.—*A* and *B*, Case 12. Gutta-percha. Observation period, five days. Cavity-pulp distance, 2.6 mm. Circulatory disturbances, accumulation of inflammatory cells. Large cavity formations partly artifacts.

B, Odontoblast nuclei in dentinal tubules, neutrophilic leukocytes in odontoblast layer. Dilated capillaries and extravasated erythrocytes.

C, Case 30. Another tooth from the same person as in *A*. Zinc oxide-eugenol sealed with gutta-percha. Observation period, five days. Some odontoblast nuclei in dentinal tubules. No other reactions.

TABLE III. LIQUID LINERS—GUTTA-PERCHA

	CASE NO.	AGE	TOOTH	DURATION (DAYS)	CAVITY-PULP DISTANCE (MM.)	ODONTOBLAST NUCLEI IN DENTINAL TUBULES	IRRITATION DENTINE	PRESENCE OF INFLAMMATORY CELLS		HYPEREMIA	OCCURRENCE OF BROWN PIGMENT	FIG. NO.
								IN ODONTOBLAST LAYER	CENTRALLY IN PULP			
A. Unifolan	34	11	5+	7	1.6	Some and erythrocytes	0	Some	Some	Slight	0	
	35	12	4−	7	1.4	Some +	+	Some and eosinophils	Some	Strong	Slight	
	36	13	4+	7	2.1	Numerous and erythrocytes	0	Some and eosinophils	Some	Moderate	0	8, *A* and *B*
B. Pulp-dent	37	11	4−	2	1.9	Few	0	0	0	0	0	
	38	11	−4	2	1.6	Few	0	0	0	Moderate	0	
	39	10	4−	5	1.9	Few	0	0	0	Slight	0	
	40	11	+4	7	1.2	Some and erythrocytes	0	Few	Few	Strong	Frequent	
	41	11	4−	9	1.8	Few and erythrocytes	+	Few	Few	Strong	Frequent	
	42	11	−4	16	1.9	0	0	0	Some	Strong	Slight	
C. Copalite	43	12	+5	5 min.	1.8	0	0	0	0	0	0	
	44	11	−4	1	1.6	Numerous and erythrocytes	0	Numerous	Numerous	Strong	Frequent	
	45	10	4−	2	1.1	Numerous	0	Numerous	Numerous	Strong	Frequent	8, *C* and *D*
D. Chloro-percha	46	10	4+	12 min.	1.4	0	0	0	0	0	0	
	47	10	4+	1	1.1	Numerous and erythrocytes	0	Some	Few	Moderate	0	
	48	12	4−	1	1.4	Some	0	Some	Few	0	0	
	49	12	4+	1	1.6	Some	0	Few	0	0	0	
	50	10	+4	1	2.1	Few	0	Few	0	0	0	
	51	10	−4	1	2.0	Few	0	0	0	0	0	
	52	9	4−	2	1.0	0	0	0	0	Moderate	0	
	53	11	+4	2	1.6	Numerous	0	Numerous	Numerous	Strong	Moderate	
	54	11	4+	7	1.3	Few	0	Few	Some	Moderate	Slight	
	55	10	4−	21	1.0	Few	+	0	0	Moderate	Slight	
	56	10	4−	23	1.5	0	+	0	Few	Moderate	0	

In those cases in which different liquid liners had been applied before the insertion of gutta-percha (Table III), a less severe reaction occurred than in the unlined cases. On the other hand, the reactions were not avoided, as clearly demonstrated in Fig. 8. In general, the following reactions were present: disturbance of circulation, migration of odontoblasts into the cut dentinal tubules, and gathering of neutrophilic leukocytes along the predentine layer (Fig. 8, *B* and *C*) and more centrally in the pulp tissue (Fig. 8, *D*).

When gutta-percha had been replaced by amalgam, the degree of healing attained varied according to the duration of the observation periods (Table IV). In cases in which the replacement had taken place a short time before the extraction, the reactions were the same, more or less, as if no replacement had taken place, whereas in cases in which there had been a relatively long observation period after the substitution a marked difference was observed. A comparison of two of these cases will demonstrate this phenomenon. In Case 59 (Table IV) after an observation period of two days the common reaction to gutta-percha was observed (Fig. 9, *A* to *C*). In Case 60 (Table IV), where gutta-percha was replaced by amalgam after two days and left there for thirty-five days, the following findings were characteristic: Odontoblast nuclei were no longer present in the dentinal tubules, but the number of odontoblasts in the adjacent layer was reduced (Fig. 9, *E*). The predentine layer formed in this area was broader than in the surrounding areas. In addition, it was more irregular and contained a reduced number of dentinal tubules, corresponding to the remaining odontoblasts (Fig. 9, *E*). Hyperemia had decreased, as the capillaries in the odontoblast layer were no longer conspicuous. Neutrophilic leukocytes were no longer observed in the odontoblast layer, but more centrally in the pulp lymphocytes and transition forms of inflammatory cells were present (Fig. 10, *C*). In other cases in the same group, plasma cells and macrophages were seen as well. In vessels located centrally in the pulp, a brown pigment was observed. It was mostly concentrated to the walls of the vessels, but it occurred inside the vessels and in their vicinity as well (Fig. 10, *A*). This brown pigment appeared to be birefringent in polarized light (Fig. 10, *B*), indicating a hemorrhage when it occurred in areas with extravasated erythrocytes and stasis when it occurred in the vessels.

DISCUSSION

It is generally accepted that it is difficult to compare the results of biologic experiments carried out and published by investigators working in the same field. This is due to the fact that they do not use the same criteria in evaluating their material. Even when evaluating the same histologic section, one investigator may consider a reaction insignificant while others will term it moderate or severe. This personal evaluation can never be avoided. Nevertheless, a comparison would be possible if the authors tabulated their observations and referred to photomicrographs demonstrating the reactions described. These photomicrographs must be taken at a magnification sufficiently large to show the

TABLE IV. REPAIR

	CASE NO.	AGE	TOOTH	DURATION (DAYS)	CAVITY-PULP DISTANCE (MM.)	ODONTOBLAST NUCLEI IN DENTINAL TUBULES	IRRITATION DENTINE	PRESENCE OF INFLAMMATORY CELLS		HYPEREMIA	OCCURRENCE OF BROWN PIGMENT	FIG. NO.
								IN ODONTOBLAST LAYER	CENTRALLY IN PULP			
A. Amalgam substituted for gutta-percha	57	10	4 –	2	0.9	Numerous	0	Numerous	Numerous	Strong	Moderate	
	58	10	– 4	2 + 2	1.2	Numerous	0	Some	Numerous	Moderate	0	
	59	12	4 +	2	2.4	Numerous	0	Numerous	Numerous	Slight	Slight	9, *A* to *C*
	60	12	+ 4	2 + 35	2.3	0	+	0	Some	Strong	Frequent	9, *D* and *E*; 10
	61	13	4 +	2	0.8	Numerous	0	Numerous	Numerous	Strong	Slight	
	62	13	+ 4	2 + 47	0.8	0	+	0	Some	Strong	Slight	
	63	10	– 4	7	1.9	Some +	0	Some	Some	Strong	Moderate	
	64	10	4 –	7 + 2	1.6	Some +	0	Some	Some	Strong	Slight	
	65	10	4 +	7 + 14	0.5	Some	+	Numerous	Numerous	Strong	Slight	
	66	10	+ 4	7 + 29	1.5	0	+	0	Some	Slight	Slight	
	67	11	– 4	7	1.9	Some + and erythrocytes	0	Few	Some	Strong	Slight	
	68	11	4 –	7 + 14	1.6	Some	0	0	Some	Slight	Slight	
	69	11	4 +	7 + 30	1.4	0	+	Few	Numerous	Strong	Moderate	
	70	11	+ 4	7 + 44	1.3	Few	+	0	Numerous	0	Slight	
	71	10	+ 4	7	1.9	Some + and erythrocytes	0	Some	Some	Strong	Frequent	
	72	10	4 +	7 + 14	0.5	0	+	0	Some	0	Moderate	
	73	10	4 +	19	0.7	Few + and erythrocytes	+	Some	Some	Strong	0	
	74	10	+ 4	19 + 9	0.7	0	+	0	Numerous	Strong	Slight	
	75	13	– 4	30 + 7	1.5	Few	+	0	Some	Strong	Slight	

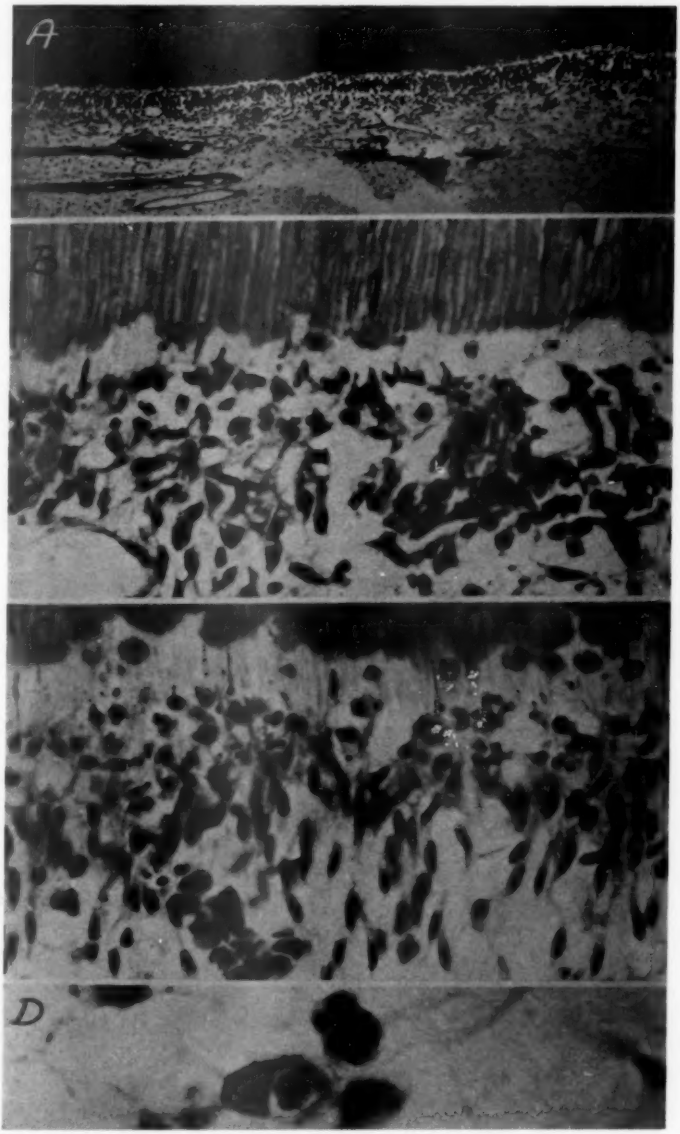

Fig. 8.—*A* and *B*, Case 36. Unifolan and gutta-percha. Observation period, seven days. Cavity-pulp distance, 2.1 mm. *A*, Circulatory disturbances, accumulation of inflammatory cells. *B*, Odontoblast nuclei in dentinal tubules, neutrophilic leukocytes and extravasated erythrocytes in odontoblast layer.

C and *D*, Case 45. Copalite and gutta-percha. Observation period, two days. Cavity-pulp distance, 1.1 mm. *C*, Odontoblast nuclei in dentinal tubules, neutrophilic leukocytes in odontoblast layer. Dilated capillaries. *D*, Neutrophilic leukocyte included in macrophage centrally in the pulp.

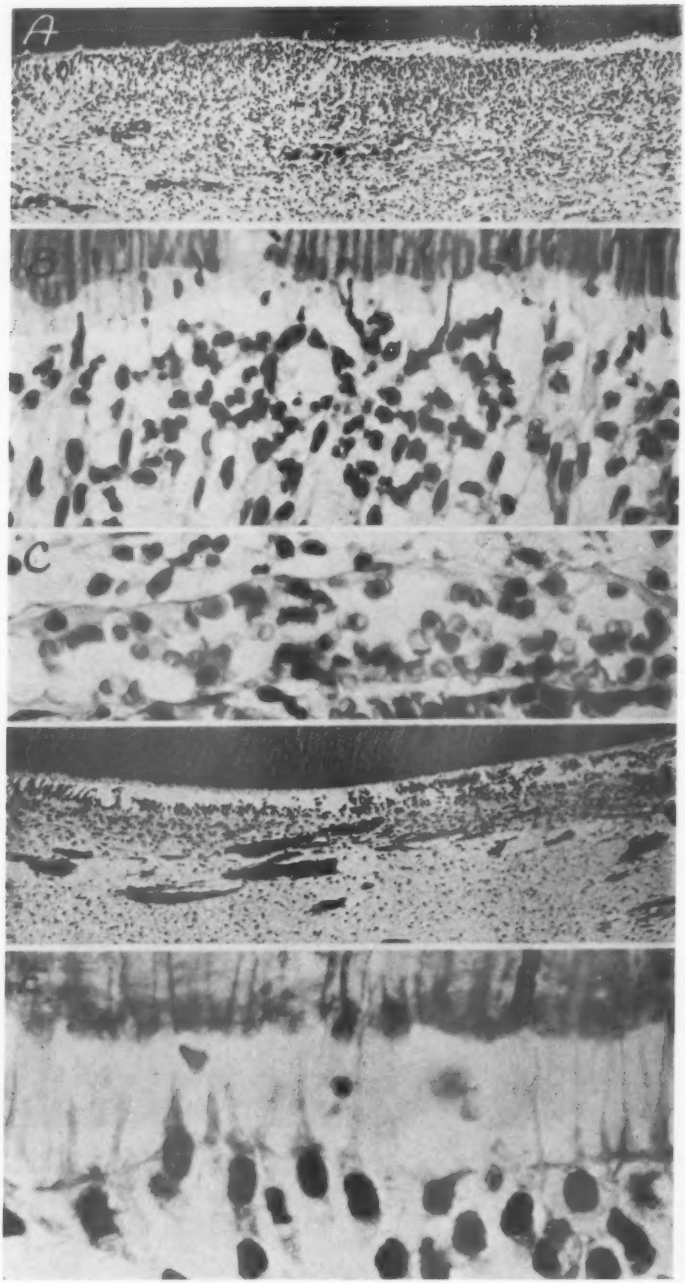

Fig. 9.—*(For legend, see opposite page.)*

details described, and the printing must be of such a quality that these details are clearly reproduced. This will necessitate a larger number of photomicrographs, but it will facilitate the discussion of a problem.

In any investigation concerning pulp reactions to filling materials, cavity preparation must be carried out in such a way that no reactions will occur as a result of the operative procedure. It has been shown experimentally that this is possible if an efficient water spray is applied during preparation.[10] It is not sufficient that the engine in question is equipped with a spray system, as shown in the experiments with different types of air rotors.[12, 13] It must be proved that the engines can be used without causing initial pulp reactions. If initial reactions are produced, as shown in Fig. 1, they will persist and develop, even if a nonirritant material has subsequently been inserted into the cavity, as shown in Fig. 2. Of course, the zinc oxide-eugenol might be blamed, at least partly, for the reactions observed in Fig. 2. However, practically no reactions occurred when the cavity was prepared with an air turbine running at 50,000 r.p.m. combined with an air-water spray before the insertion of zinc oxide-eugenol (Fig. 3, *A*). The slight reaction observed (that is, an insignificant formation of irritation dentine in a circumscribed area under the cavity) indicates that some odontoblasts have deteriorated without causing a chronic inflammatory reaction in the pulp. Accordingly, the preparation method used may be one of the reasons why zinc oxide-eugenol, or its components, has been blamed for pathologic pulp reactions in some previous publications. It should be stressed that the zinc oxide-eugenol used in the present experimental series contains pure chemicals only and is free of foreign matter. Thus, the method of cavity preparation is the cause of the pathologic reactions demonstrated in Fig. 2.

According to the clinical result of cavity preparation, one more factor should be pointed out. It is obvious that the heat produced locally by any kind of rotating instrument may be sufficiently high to damage tooth structure. If this happens at the margins of a cavity, it may be one of the most important origins of secondary or recurrent caries.

It has been shown that cavity preparation under air spray results in migration of odontoblast nuclei into the dentinal tubules. One must therefore assume that this reaction has taken place initially in the experiments of James and Schour,[7] even if those investigators did not report such a finding. Half of their experiments were performed with an airbrasive, by which a continuous stream of aluminum oxide and air is directed to the field of operation. This would result in a desiccation of tooth substance, causing odontoblast nuclei to migrate into the dentinal tubules.[10] Because of the subsequent deterioration of the migrated odontoblasts, neutrophilic leukocytes would accumulate in the

Fig. 9.—*A*, *B*, and *C*, Case 59. Gutta-percha. Observation period, two days. Cavity-pulp distance, 2.4 mm. *A*, Accumulation of inflammatory cells. *B*, Odontoblast nuclei in dentinal tubules, neutrophilic leukocytes in odontoblast layer. *C*, Neutrophilic leukocytes inside and outside vessels centrally in the pulp.

D and *E*, Case 60. Gutta-percha replaced by amalgam after two days, amalgam left in cavity thirty-five days before extraction. *D*, Circulatory disturbances. Pulp tissue partly torn from dentine. *E*, Broad predentine layer containing fewer and more irregular dentinal tubules than dentine formed before experiment. Number of odontoblasts reduced. Remaining odontoblasts contain scattered chromatin and nucleoli.

adjacent pulp tissue. Accordingly, James and her co-workers[5] cannot ascribe their findings to gutta-percha exclusively but to the mixed influence of the cavity preparation method in question and the gutta-percha subsequently inserted into the cavity. This would also be the case in Barker's[4] experiments, even though Barker drew the clinical conclusion that it was safe to prepare cavities by applying strong pressure to a worn burr and drying the cavities thoroughly with hot air before the gutta-percha was inserted. In Rovelstad and St. John's[3] experiments the cavities were exposed to sodium fluoride and then sealed with gutta-percha. Thus, these authors also recorded a mixed reaction, namely, the possible reaction to sodium fluoride and the reaction to gutta-percha, which at that time was known through the previous publication of Hellner.[1]

None of the authors writing about reactions to gutta-percha have reported the observation of odontoblast nuclei in the dentinal tubules. This may be a result of the histologic method used. They would appear in thin paraffin sections (about 4 to 5 microns), and they were a constant finding in all cases of the present experimental series.

In other publications, however, the occurrence of odontoblast nuclei in the dentinal tubules is considered an important criterion of a pulp reaction, but the explanation of this migration is varying. Kramer and McLean[14] believed that the odontoblasts were sucked in with the tissue fluid which returned to the dentinal tubules after being pressed into the pulp as a result of a temperature increase caused by the polymerization of acrylic fillings. Stanley and Swerdlow,[15] in a contradictory explanation, have maintained that the cells were pressed into the tubules in response to an increased pulp pressure caused by inflammation. None of these explanations seem adequate. First and foremost, it does not seem possible that a large body like the odontoblast could be pressed into a narrow dentinal tubule. In addition, this reaction may take place within two seconds,[10] a time period considered too short for an inflammation to cause an increase of the pressure in the pulp. Furthermore, this pressure would spread in all directions, and the odontoblast nuclei are observed exclusively in the cut dentinal tubules. Most of the evidences previously published seem to indicate that the odontoblasts are sucked into the dentinal tubules because of an increase of the osmotic pressure in the peripheral part of the cut odontoblasts.[10]

The statement by Stanley and Swerdlow[16] that "tenths of millimeters can change greatly the outcome of an experiment" is not corroborated by the present experimental series. On the contrary, a study of the tables shows in more than one case a slighter reaction under a deeper cavity (for example, Cases 28 to 30, Figs. 6, *D* and 7, *C*). In addition, tenths of millimeters can hardly be measured with accuracy, because the dentinal tubules are curved and the measuring part of a micrometer ocular is straight.

Various authors disagree as to the broadness of the predentine layer. In the present experimental series, an increase in predentine formation seemed to be prevalent in most cases. The explanation is that the odontoblasts which had migrated into the dentinal tubules were those located nearest the predentine before the filling was inserted. Thus, the remaining odontoblasts located most

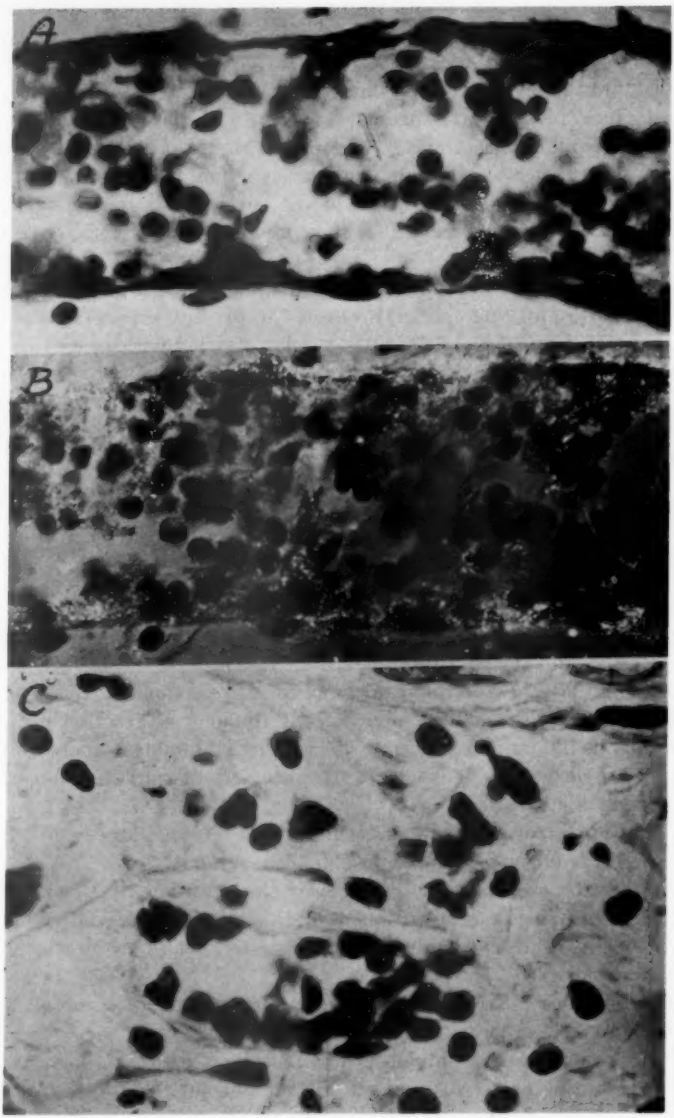

Fig. 10.—Case 60 (continued).

A, Vessel containing erythrocytes and scattered brown pigment. Brown pigment concentrated to vessel wall.

B, Same vessel as in *A*. Photomicrograph taken in polarized light shows birefringency of brown pigment.

C, Lymphocytes centrally in pulp tissue.

centrally in the pulp will be those bordering the predentine in this area (Fig. 2, *B*). This will give the impression of an increase in the rate of predentine formation.

Contrary to the observation by James and co-workers[5] no new formation of odontoblasts took place in the present experimental series. Cells resembling those termed preodontoblasts and young odontoblasts have been observed, but they are considered part of the inflammatory reaction, especially since the final result in all experiments in which part of the odontoblasts have deteriorated is usually a layer of irregular dentine and predentine bordered by a reduced odontoblast layer.

Margin leakage and too high temperature have been considered the cause of the reactions to gutta-percha.[6] However, the present experiments show that the reactions still occur even when gutta-percha is sealed with zinc oxide-eugenol or amalgam. On the other hand, no reactions occurred when zinc oxide-eugenol was used as a liner. Furthermore, the method of inserting the gutta-percha in these experiments would not give any temperature rise. Thus, the reaction to gutta-percha seems to be connected with the material itself.

SUMMARY AND CONCLUSIONS

1. Cavity preparation may be carried out without causing reactions in the pulp.

2. If the water spray of the engine in question is insufficient, odontoblast nuclei will migrate into the cut dentinal tubules; if zinc oxide-eugenol is subsequently inserted into the cavity, an acute inflammatory reaction will occur and under some circumstances develop into a chronic inflammatory reaction.

3. Insertion of gutta-percha in cavities in the dentine resulted in inflammatory reactions in the pulp tissue, characterized by odontoblast nuclei in the dentinal tubules, neutrophilic leukocytes, lymphocytes, plasma cells, macrophages, and circulatory disturbances.

4. Using gutta-percha as an underfilling and closing the cavity with amalgam or zinc oxide-eugenol resulted in similar reactions.

5. Application of a liquid liner (Unifolan, Pulpdent, Copalite, chloropercha) would provide a certain protection, but this is not considered sufficient.

6. A thick layer of zinc oxide-eugenol was a reliable liner, by which harmful effects were avoided.

7. There was no difference in pulp reactions following the use of base plate gutta-percha or of temporary stopping.

8. New formation of odontoblasts was never observed.

9. Regeneration of the injured pulp tissue did not take place.

10. The acute inflammatory reaction subsided and left a more or less extended scar tissue and a chronic inflammation.

REFERENCES

1. Hellner, E.: Undersökningen över reaktiva förändringar i tandens pulpavävnad vid användning av olika s.k. isoleringsmedel och fyllnadsmaterial, Svenska tandl. tidskr. **21**: 225, 1928.

2. Gurley, W. B., and van Huysen, G.: Histologic Response of the Teeth of Dogs to Operative Procedures, J. D. Res. **19**: 179, 1940.
3. Hovelstad, G. H., and St. John, W. E.: Condition of the Young Dental Pulp After the Application of Sodium Fluoride to Freshly Cut Dentin, J. Am. Dent. A. **39**: 670, 1949.
4. Barker, J. N.: Reactions of the Pulp to Various Filling Materials and Clinical Procedures, Australian J. Dent. **46**: 226, 1942.
5. James, V. E., Schour, I., and Spence, J. M.: Response of Human Pulp to Gutta-Percha and Cavity Preparation, J. Am. Dent. A. **49**: 639, 1954.
6. Massler, M.: 1958: Pulp Protection and Preservation, Chicago, 1958, Year Book Publishers, Inc., p. 21.
7. James, V. E., and Schour, I.: Effect of Cavity Preparation Alone on the Human Pulp, J. D. Res. **34**: 758, 1955.
8. Norlén, I.: Air-driven Dental Handpiece With Manual Speed Control: "Dentalair Unit," Acta odont. scandinav. **15**: 199, 1957.
9. Östby, B. N.: A Manual in Endodontics, Oslo, 1958, The Norwegian Institute of Dental Research, p. 28.
10. Langeland, K.: Tissue Changes in the Dental Pulp, Odontol. Tidskr. **65**: 243, 1957.
11. Langeland, K.: En sammenligning av pulpareaksjoner på low og high speed preparering, og om pulpareaksjoner på fyllingsmaterialet Cavinyl, Norske tannl. tid. **66**: 994, 1956.
12. Langeland, K.: Pulp Reactions to High-Speed Instrumentation, Norske tannl. tid. **68**: 137, 1958.
13. Östby, B. N.: Clinical and Experimental Experience With Borden Airotor, Norske tannl. tid. **68**: 135, 1958.
14. Kramer, J. R. H., and McLean, J. W.: The Response of the Human Pulp to Self-Polymerizing Acrylic Restorations, Brit. Dent. J. **92**: 255, 1952.
15. Stanley, H. R., Jr., and Swerdlow, H.: Aspiration of Cells Into Dentinal Tubules? Oral Surg., Oral Med. & Oral Path. **11**: 1007, 1958.
16. Stanley, H. R., Jr., and Swerdlow, H.: Reaction of the Human Pulp to Cavity Preparation: Results Produced by Eight Different Operative Grinding Technics, J. Am. Dent. A. **58**: 49, 1959.

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ARMED FORCES AND PUBLIC HEALTH SERVICE

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BILATERAL TRAUMATIC FACIAL PARALYSIS

Report of a Case

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THIS report concerns an interesting neuropathy associated with fractures of the skull and facial bones. The case to be presented was unusual in that several cranial nerves were bilaterally involved over prolonged periods of time. The patient, however, had no demonstrable meningeal injury and presented only such symptoms as were directly related to the neuropathy. The obvious, outstanding findings were (1) the completely "frozen" face of bilateral seventh cranial nerve paralysis (Bell's palsy), (2) eighth cranial nerve paresis with total loss of hearing on one side and subtotal loss on the other, and (3) bizarre ocular movements associated with bilateral sixth cranial nerve involvement. Other less dramatic symptoms will be subsequently noted in this context. This case may be considered a clinical rarity from two standpoints: (1) the patient survived the traumatic incident and (2) investigation of the literature revealed only one case of bilateral facial paralysis reported since 1930.[1]

CASE REPORT

On Dec. 30, 1959, a 24-year-old Caucasian male soldier was admitted to the oral surgery service of the U. S. Naval Hospital at Great Lakes, Illinois, with a posttraumatic jaw and facial bone fracture problem and concomitant neuropathy involving several cranial nerves. On Nov. 25, 1959, the patient was participating in military maneuvers in Germany when the accordion roof of the mortar truck in which he was riding closed on his head, producing the fairly typical "pumpkin crush" type of injury. As an immediate result of this trauma, the patient was unconscious for an undetermined period of time and was admitted to a military hospital in Germany where he received emergency care. Multiple lacerated wounds of the left face and the right mastoid area were treated, and surgical reduction

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The opinions or assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large or the Department of the Air Force or the Air Service at large.

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of the fractured left zygomatic complex was performed. In addition to the facial trauma, bilateral hemotympanum was noted and cerebrospinal otorrhea was suspected. The patient was transferred to this Naval Regional Hospital approximately five weeks after he sustained the injury.

The past and family histories were considered to be noncontributory.

Physical examination revealed a well-developed, well-nourished young man who was ambulatory and in no acute distress. He was well oriented except for events immediately following his injury. There were multiple healed facial and scalp lacerations. The injured left zygoma was firm and in good position. Three startling objective findings were readily apparent: (1) disoriented, extraocular movements with total lack of amblyopic function, (2) complete inability to move any of the muscles of expression, and (3) marked loss of hearing in the right ear and moderate decrease of auditory acuity in the left ear. The patient's speech had the quality of that of a deaf-mute, further complicated by the loss of function of the facial muscles. The patient was completely edentulous, having lost his artificial dentures in the accident. (Figs. 1 and 2.)

Fig. 1. Fig. 2.

Fig. 1.—The complete loss of function of all the muscles of expression is demonstrated. The patient is exerting maximum effort to contract his facial muscles. Note also the convergent strabismus.

Fig. 2.—Mouth opening and closing, a function of the fifth rather than the seventh cranial nerves, was unaffected. However, with the seventh cranial nerves involved bilaterally, note the loss of tone and function of the facial muscles. The eye patch served a dual purpose: (1) it eliminated the annoyance of diplopia and (2) it afforded protection since palpebral function was largely absent. The patch was worn alternately on one eye and then the other.

The admission laboratory findings were reported to be within normal limits.

Skull roentgenograms recorded the comminuted fracture of the left zygomatic complex which had been well reduced. Repeated films failed to demonstrate evidence of skull fracture, although neurological findings supported the probability of such a fracture.

Consultations by the otolaryngological, ophthalmological, neurological, and neurosurgical services were obtained and were recorded as follows:

Otolaryngological examination revealed both ear drums to be intact, but with hemotympanum on the left side. Bell's phenomenon was noted bilaterally. Audiograms established a 48 decibel loss on the right side and a 22 decibel loss on the left. A repeat audiogram two months later revealed no improvement on the right side and the loss of an additional 5 decibel in the left ear.

Ophthalmological examination revealed VOD 20/40 and VOS 20/20. The funduscopic examination was normal, and the visual fields were within normal limits. Anisocoria was evident, with the right pupil the largest. Each pupil reacted to direct and consensual light, although the right pupil was sluggish. There was paralysis of the right external rectus muscle and paresis of the left external rectus muscle.

Neurological examination revealed that the patient's mental status was normal, although his speech was that of a deaf-mute. Olfactory sensation was functional. Taste

Fig. 3. Fig. 4.

Fig. 5.

Fig. 3.—Three months after injury. Note the return of function of the middle and lower branches of the seventh cranial nerves. The frontal branch, as anticipated in such cases, was the last to regain function.

Fig. 4.—The return of muscular function was from inferior to superior. First, the commissures of the mouth regained tonus and sphincter activity. Then the nasolabial folds became apparent. Subsequently, palpebral activity was re-established. Ocular motions are still somewhat bizarre.

Fig. 5.—This effort at whistling represents a considerable return of function of the facial nerves. There is also a progressive improvement in the convergent strabismus.

sensation was apparently lacking, or greatly diminished, over the distribution of the lingual nerve. In those areas supplied by the ninth cranial nerve, taste sensation was apparently normal. The visual acuity was good, and the visual fields and fundi were normal. Extraocular movements revealed a bilateral sixth cranial nerve paralysis, with convergent strabismus, and an alternating fixation of vision. There was bilateral paralysis of the seventh cranial nerves with a complete masklike appearance to the face. On repeat evaluation two months later, a slight return of function of the lower branches of the seventh nerves was observed, most noticeable at the nasolabial folds (Figs. 3, 4, and 5.) There was also bilateral involvement of the eighth cranial nerves, with complete deafness on the right side and approximately 50 per cent loss of hearing in the left ear.

Neurosurgical findings concurred with those previously reported by the neurologist. It was agreed that neurosurgical intervention would be of highly doubtful benefit and that only an elapse of time could prognosticate the anticipated degree of functional improvement. It appeared evident that the injuries to the peripheral cranial nerves were at the level of the base of the skull.

The oral surgery service provided supportive care, which included vitamin B_{12} therapy and a physical medicine program concerned with galvanic current stimulation to the facial muscles. Full maxillary and mandibular denture prostheses were provided, and these produced some improvement in phonetics. The patient was hospitalized here for approximately four months. During this time there was a gradual, although slow, return of the function of the sixth and seventh cranial nerves, bilaterally. It is anticipated that this improvement will progress, although the chance for full recovery is conjectural. In contradistinction, the eighth cranial nerve has appeared to undergo further degeneration. No improvement in auditory acuity is anticipated.

DISCUSSION

Midfacial fractures and associated unilateral neuropathy are not uncommon traumatic problems of the present day. Unilateral paralysis of the facial nerve is often associated with surgical injury, infection, and idiopathic episodes. However, bilateral involvement of the sixth, seventh, and eighth cranial nerves is rare in patients who survive such injury.

The sixth and seventh cranial nerves are basically motor fibers, with the nervus intermedius comprising the sensory component of the seventh nerve. On the other hand, the eighth cranial nerve is wholly sensory, with two distinct components—the cochlear and the vestibular.[2]

The sixth cranial nerve emerges from the brain at the junction of the pyramid and the pons. It does not penetrate the dura until it reaches the dorsum sella of the sphenoid bone. It continues its long and exposed cranial route through the cavernous sinus and the superior orbital fissure to supply the lateral rectus muscle of the eye. Some authors believe that this route readily predisposes the abducens nerve to trauma associated with basal skull fracture.

The seventh and eighth cranial nerves pierce the dura near the internal acoustic meatus and, traveling together, enter this protective bony canal of the temporal bone. However, their routes are slightly different. The cochlear and vestibular nerves travel the short distance to their respective sensory sites. At the base of the meatus, the facial nerve enters the facial canal which it traverses to exit at the stylomastoid foramen. The facial nerve supplies the muscles of the face, scalp, and ear, the buccinator, platysma, and stylohyoideus muscles,

the posterior belly of the digastric muscle, and the muscle of the stapes. The sensory fibers serve the taste function of the anterior two-thirds of the tongue and carry vasodilator and secretory fibers to the submaxillary and sublingual glands.

It seems reasonable to assume that the neural injuries of this case were peripheral, since the patient maintained good vital signs and was alert and ambulatory except for a brief episode of concussion immediately following the accident. The possibility of a fracture of the internal auditory meatus must be considered, although disturbance of equilibrium was at no time a clinical problem with this patient. The closest approximation of the abducens, subdurally, to the internal auditory meatus is at the medial end of this canal. Thus, anatomically, to interfere bilaterally with these three cranial nerves in a peripheral site, a transverse basal skull fracture would probably involve the temporal bone and the occipital bone or spheno-occipital articulation. Skull fractures at the foramina-weakened base of the skull may be difficult to demonstrate roentgenographically. Without displacement of fractured bones in this area, the roentgenographic diagnosis becomes even more difficult. The fact remains, however, that symptomatology in this case supported the evidence of peripheral cranial nerve injuries at the level of the base of the skull.

SUMMARY

A case of facial bone fracture with unusual associated neuropathy has been presented. Bilateral involvement of the sixth, seventh, and eighth cranial nerves was demonstrated following a traumatic injury to the head and face.

REFERENCES

1. Costantini, H., and Curtillet, E.: Paralysie faciale bilaterale; anastomose spino-faciale et resection du ganglion cervical superieur des deux cotes, Lyon Chir. 32: 291-305, 1935.
2. Goss, C. M.: Gray's Anatomy, ed. 27, Philadelphia, 1959, Lea & Febiger, pp. 951-997.

PERIAPICAL REPAIR BY DENSE FIBROUS CONNECTIVE TISSUE FOLLOWING CONSERVATIVE ENDODONTIC THERAPY

*E. C. Penick, Lieutenant Commander (DC) USN**

THE chief criterion for success in conservative root canal therapy performed on a tooth with a roentgenolucent area in the periapical region is complete bone regeneration of the region, with re-establishment of the lamina dura and periodontal membrane. Following placement of the root canal filling, therefore, periodic checkup roentgenograms should be taken to ascertain whether periapical healing is taking place. Such healing usually occurs, provided the periapical area is not cystic, an aseptic technique was followed, and each canal was sterile and hermetically sealed. The time required for complete bone repair varies with the size of the roentgenolucent area, the age and the health of the patient, and the amount of occlusal stress to which the tooth is subjected. In most instances there is some evidence of bone repair within a few months following conservative root canal treatment and considerable evidence of regeneration within one year.

If, after a reasonable period of time (six months to a year, depending upon the circumstances), a periapical roentgenolucent area shows no roentgenographic evidence of repair, periapical curettage is indicated, provided it can be carried out without injuring other structures. This report deals with cases of this nature. In ten instances periapical curettage was performed on teeth that had yielded two negative cultures and were well filled apically and laterally but showed no evidence of bone regeneration after a minimum of six months. The periapical tissue removed during curettage was submitted for histologic study. Microscopic examination of the tissue showed four of the ten roentgenolucent areas to be radicular cysts, three to be granulomas, and the remaining three to be dense fibrous connective tissue (scar tissue).

The case presented here is typical of the cases that healed by the formation of dense fibrous connective tissue. The minimum interval of time between obturation of the canal and curettage of the roentgenolucent area was ten months for the periapical areas that had healed by fibrous tissue formation.

The opinions or assertions contained herein are the private ones of the author and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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CASE REPORT

The patient, a healthy 28-year-old man, complained of a "gum boil in the upper right side, which drains periodically." Roentgenograms revealed a roentgenolucent area involving the apex of the right maxillary lateral incisor (Fig. 1). The area was not sharply circumscribed. A draining sinus tract was present. The diagnosis was recorded as a chronic alveolar abscess associated with the pulpless lateral incisor. Conservative root canal therapy was performed on the involved tooth, following which roentgenograms of the periapical region were taken periodically to ascertain whether the lesion was healing.

Fig. 1.—Roentgenogram taken prior to treatment of right maxillary lateral incisor.

Fig. 2.

Fig. 3.

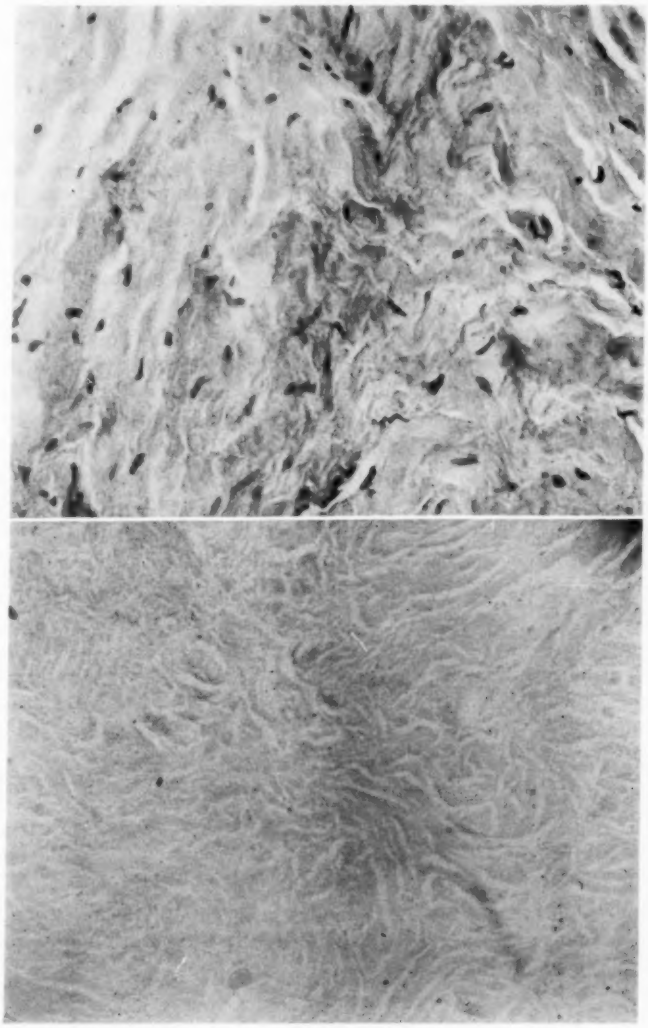
Fig. 2.—Roentgenogram taken immediately after placement of root canal filling.
Fig. 3.—Checkup roentgenogram taken fourteen months after placement of root canal filling.

The root canal was treated in three sessions. The original culture, taken at the first session, was positive. The canal was enlarged, and cultures were obtained at both the second and the third sessions. These cultures were negative. At a fourth session the root canal was filled with gutta-percha and pulp-canal sealer (Fig. 2).

The tooth was completely asymptomatic following placement of the root canal filling. Follow-up roentgenograms showed no regeneration of bone in the periapical region, but the roentgenolucent area did not increase in size. After fourteen months (Fig. 3) it was decided that a periapical curettage should be performed. A semilunar incision was made, and a mucoperiosteal flap was reflected in the usual manner. When the periosteum was reflected, it became apparent that the periosteum and the cortical plate overlying the

periapical area had been destroyed and the periapical tissue had become firmly attached to the overlying mucosa. This necessitated dissection of the periapical tissue from the mucosa. The periapical tissue was removed in toto and fixed in a 10 per cent solution of

A.



B.

Fig. 4.—Photomicrographs of avascular fibrous connective tissue removed during periapical curettage. *A*, Low-power magnification. *B*, High-power magnification from same section of tissue.

formalin. Histologic examination of the specimen (Fig. 4, *A* and *B*) showed it to be composed of dense, relatively avascular, fibrous connective tissue which showed no evidence of any inflammatory reaction.

DISCUSSION

In the reparative phase of inflammation, the body attempts to restore injured or lost tissue with tissue of the same type. The more highly specialized the tissue, the more difficult it is to restore, and in some instances a defect may be repaired with a less highly specialized tissue. This explains why any bone defect may be repaired with dense fibrous connective tissue. Leriche and Policard[1] mention healing with dense fibrous connective tissue as a possibility in bone lesions. Grossman[2] also mentions healing by fibrous tissue formation following endodontic treatment of pulpless teeth with periapical lesions, and Kukidome[3] has demonstrated this type of periapical healing.

In the case presented here, the periapical lesion had been completely replaced by healthy, dense, fibrous connective tissue. This tissue was free from inflammatory cells and could not possibly serve as a focus of infection or deplete the defense potential of the body. Therefore, periapical curettage was unnecessary.

The three lesions that healed by formation of scar tissue had certain similarities: All had sinus tracts at the time root canal treatment was initiated, and all were diagnosed clinically as chronic alveolar abscesses. These lesions were thought to have existed for long periods of time prior to endodontic treatment of the associated pulpless teeth. The initial cultures from the root canals of these teeth were positive, and the overlying periosteum and cortical plate of bone had been destroyed in each instance. It would appear that in these cases destruction of the overlying cortical plate of bone and periosteum must have occurred prior to endodontic treatment of the associated tooth to permit an ingrowth of fibrous tissue from the overlying mucosa during the healing process.

Although data derived from ten cases cannot be considered statistically significant, it is quite possible that periapical healing by formation of scar tissue occurs more frequently than we suspect. The periapical area remains roentgenolucent in such cases, and it is not known how this condition may be distinguished from an asymptomatic unhealed periapical lesion.

CONCLUSIONS

The possibility of healing by formation of scar tissue should be borne in mind when periapical curettage is being considered in those cases in which bone regeneration has not occurred after a reasonable length of time following conservative root canal therapy. Further study of the etiology and means of diagnosis of this condition is needed.

REFERENCES

1. Leriche, R., and Policard, A.: The Normal and Pathologic Physiology of Bone, St. Louis, 1928, The C. V. Mosby Company, p. 17.
2. Grossman, L. I.: Root Canal Therapy, ed. 4, Philadelphia, 1955, Lea & Febiger, p. 341.
3. Kukidome, K.: Histopathological Study on Healing of Periapical Tissues After Infected Root Canal Treatment of Human Teeth, Bull. Oral Path., Tokyo 2: 65, 1957.

CYST OF THE GINGIVA (MUCOCELE)

Report of a Case

Kimble A. Traeger, Commander (DC), USN

CYSTS of the gingiva have been reported infrequently. Gingival cysts with clinical manifestations are apparently rare, and no reference to a mucocele of the gingiva could be found in the literature. Ritchey and Orban[1] reported six cysts of the gingiva observed in the histologic examination of 350 gingival biopsy specimens; however, the cysts were not detected macroscopically. Bhaskar and Laskin[2] reported three cases of gingival cysts which were discernible clinically and roentgenographically, but no glandular elements were found in the area and it seems unlikely that these cysts were of glandular origin.

The case reported here involved a cyst of the attached gingiva which, on histologic examination, was found to be associated with heterotopic glandular elements of the gingiva.

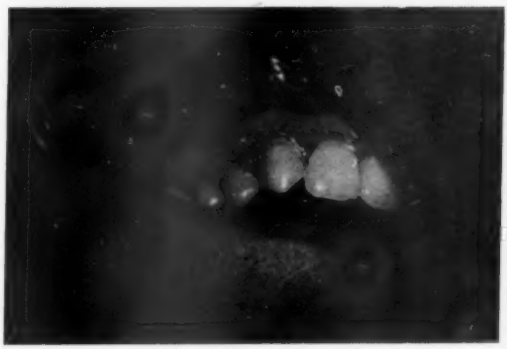


Fig. 1.—Cystic lesion on the attached gingiva between cuspid and premolar.

CASE REPORT

Routine dental examination of a 19-year-old Caucasian Marine recruit revealed a cystic lesion on the facial aspect of the attached gingiva between the maxillary right cuspid and first premolar (Fig. 1). The lesion was a sharply circumscribed, elevated, smooth, shiny

From the United States Marine Corps Recruit Depot, San Diego, California.

The opinions or assertions contained in this article are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

fluctuant mass. There was no evidence of infection or inflammation, and no history of trauma was elicited. The mass was nontender and asymptomatic. Since the patient was not aware of its existence, the duration of the lesion was undetermined. The gingiva and oral tissues in other respects appeared to be normal. Roentgenographic examination revealed no bone involvement.

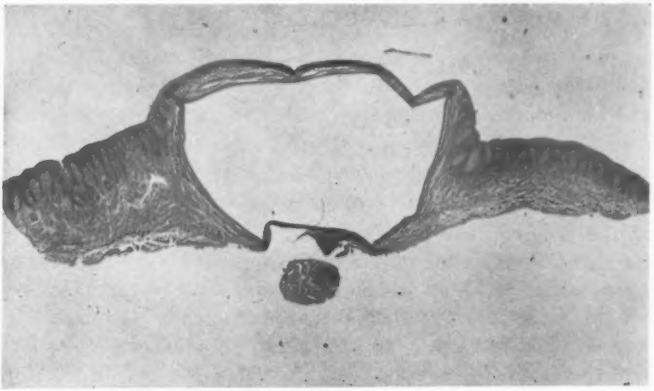


Fig. 2.—Topographic view of the gingival cyst. Inferior to the cystic area there is a small mass of salivary gland tissue.

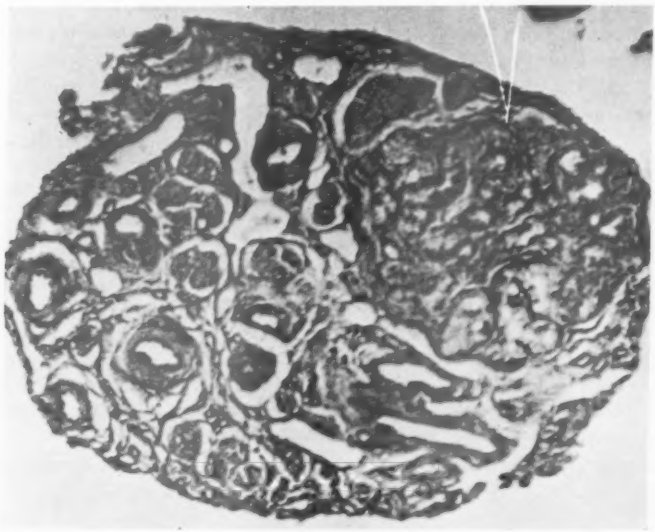


Fig. 3.—Salivary gland acini, small nerves, and vessels which were adjacent to the inferior margin of the cystic space.

The lesion was observed periodically for two weeks, with no apparent change. It was then excised and submitted for histologic examination.

Microscopic examination was reported as follows: "Examination reveals sections of mucous membrane in the center of which the mucosal surface is slightly but rather abruptly elevated above the normal level (Fig. 2). The epithelium overlying this area is very thin, and

immediately beneath the epithelium there is a large, clear space lined by a single layer of compressed epithelial cells. Adjacent to the inferior margin of this space there is a fragment of tissue composed of salivary gland acini, small nerves, and vessels (Fig. 3). The specimen could represent a mucocele. However, the fluid material was lost from the space during the processing of the tissue, and special staining fails to reveal even traces of mucin within the space."

DISCUSSION

The clinical and histologic findings in this case strongly suggest that this lesion was a mucocele of the gingiva. Mucoceles are seen frequently on the lower lip and less frequently on the buccal mucosa, palate, floor of the mouth, retromolar region, and other miscellaneous locations.[3] These are regions where mucous glands are normally found. Glands do not appear normally in the gingiva, but their presence in the gingiva has previously been reported.[1]

Ritchey and Orban[1] state that the origin of glands in the gingiva can be explained only on the basis of developmental displacement and that it is possible that such heterotopic glandular elements might undergo cystic transformation and give rise to small cysts.

In the case reported here there is no evidence of cystic transformation of glandular elements, but the histologic picture is that of a mucocele showing glandular elements inferior to a retention cyst. Even though the presence of mucin was not proved in this specimen, the evidence seems conclusive that this cyst was, in fact, a retention cyst or mucocele of the gingiva.

SUMMARY

A case of clinically discernible cyst of the attached gingiva has been reported. Histologic examination of the lesion showed the presence of heterotopic glandular elements and the formation of a cyst that was similar in all respects to a mucocele.

REFERENCES

1. Ritchey, B., and Orban, B.: Cysts of the Gingiva, ORAL SURG., ORAL MED. & ORAL PATH. 6: 765, 1953.
2. Bhaskar, S. N., and Laskin, D. M.: Gingival Cysts, ORAL SURG., ORAL MED. & ORAL PATH. 8: 803, 1955.
3. Standish, S. M., and Shafer, W. G.: The Mucus Retention Phenomenon, J. Oral Surg. 17: 15, 1959.

FRACTURES OF THE MAXILLARY TUBEROSITY OCCURRING DURING TOOTH EXTRACTION INVOLVING THE MAXILLARY ANTRUM

Jack Traiger, Captain, DC, USA

OCCASIONALLY during the course of extraction of the maxillary second or third molar, the maxillary tuberosity may be fractured and can be felt to be moving with the forceps. When this happens, the problem is whether or not to proceed with the extraction. This is important since the tuberosity and the bone over the molar area form part of the floor of the maxillary sinus, and removal produces a perforation of appreciable dimension. The predisposing causes of a fractured maxillary tuberosity are a large maxillary sinus with thin walls, large divergent roots, an abnormal number of roots, or ankylosis. Chronic periapical infection also tends to weaken the bone, thereby making the bone of the tuberosity more liable to fracture.

When the tooth is being extracted because of pain, the operator has only one choice—to remove the molar together with the attached tuberosity. When the maxillary tuberosity bears only one tooth, an incision should be made at once along the crest of the tuberosity from as far back as possible to the gingival margin of the tooth, so that a flap can be turned both buccally and palatally to expose the fractured section. After all soft tissue is completely detached, the bone segment with the tooth is carefully dissected out. In order to avoid introducing infection, no attempt is made to disturb or aspirate the blood clot within the sinus. The bone wound is débrided, the gingival margins are trimmed so that they can be approximated properly, and the wound is closed by continuous or interrupted sutures. An acrylic stent may be constructed to support the wound margins, but healing will occur without it. The patient is given a five-day course of intramuscular penicillin to prevent infection on the affected side. The patient is warned not to do excessive rinsing and not to blow his nose for at least one week. The patient should also be warned that there may be slight bleeding from the nostril on the affected side for at least a day or two. It is important that the patient be seen by an ear, nose, and throat specialist who will be able to deal with any possible infection of the sinus. It is also advisable that postoperative roentgenograms of the affected sinus be taken after two months to see whether or not healing is progressing satisfactorily.

If fracture of the tuberosity occurs during extraction of a tooth that gives no pain but has to be removed for construction of a prosthesis, the tooth may

From the Oral Surgery Section, Post Dental Clinic, Fort Jay, Governors Island, New York, New York.

be retained for a few months and then be removed surgically. Its antagonist in the mandible may be removed to prevent pain on occlusion, or the tooth can be splinted. The patient is given a five-day course of intramuscular penicillin to prevent infection.

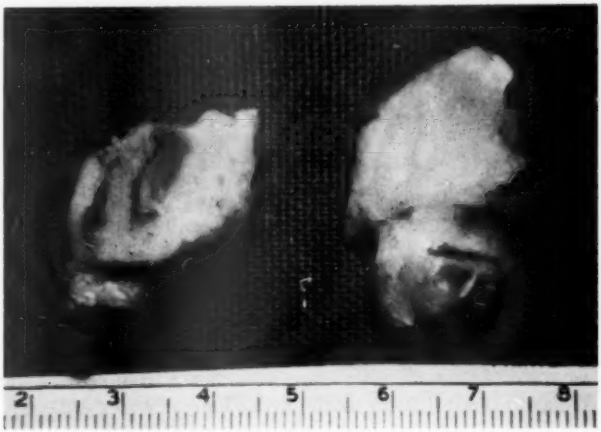


Fig. 1.

Fig. 2.

CASE REPORTS

CASE 1.—A 20-year-old man presented for extraction of a painful upper left second molar under local anesthesia (2 per cent Xylocaine hydrochloride). While the tooth was being extracted, the whole of the tuberosity was felt to move. An incision was made on the buccal aspect of the upper left second molar, beginning in the mucobuccal fold and extending to the mesial aspect of the tooth. The incision was continued around the gingival margin and along the crest of the tuberosity. The mucoperiosteal flap was reflected and the detached tuberosity was carefully dissected out. The incision was closed with interrupted sutures, and the patient

was given penicillin intramuscularly for five days. After ten days the sutures were removed. The patient was seen by the ear, nose, and throat surgeon for follow-up. Healing was uneventful. Figs 1 and 2 show the tuberosity after removal.

CASE 2.—A 31-year-old man presented for extraction of a painful upper left second molar under local anesthesia. During removal of the tooth, something was felt to be mobile. A buccal mucoperiosteal flap was raised, and the tooth and a large segment of the bone which formed the wall of the maxillary sinus were dissected out. The incision was closed with interrupted sutures, and the patient was placed on intramuscular penicillin for five days. Healing was uneventful, and follow-up roentgenograms revealed a normal sinus. Figs. 1 and 2 show the sinus wall after removal.

DISCUSSION

The removal of a tuberosity or other large bony defect will increase the difficulty of fitting a prosthesis at some future date, but the problem is one which eventually can be corrected. In both cases presented here the surgical procedure was completed because of pain in the maxillary molars.

SUMMARY

Two cases are described in which maxillary sinus involvement and tuberosity fracture were encountered during tooth extraction.

ANNOUNCEMENTS

Spanish Society of Orthodontics

The seventh meeting of the Society will be held April 27 to 30, 1961, in Valencia, Spain, under the chairmanship of A. J. Cervera, Av. del Oeste, 29, Valencia. The secretary is S. Alvarez Biosca, Av. José Antonio, 62, Madrid.

Waldemar Medical Research Foundation

Establishment of a dental clinical pathology group for oral diseases at the Waldemar Medical Research Foundation has been announced by Dr. Norman Molomut, scientific director of the Port Washington research organization. In describing the activities of the new group, Dr. Molomut also announced the appointment of Dr. Paul Scheman, prominent dental surgeon, as senior research scientist.

The Waldemar Medical Research Foundation is located at 16 Sintsink Drive East, Port Washington, New York.

Jewish Chronic Disease Hospital

Applications are being received for a one-year residency in anesthesiology for graduate dentists under Stanley R. Spiro, D.D.S., at the Jewish Chronic Disease Hospital, beginning July 1, 1961. Full maintenance and a stipend of $200.00 per month are provided.

Requests should be addressed to the Director of Dentistry, Jewish Chronic Disease Hospital, 86 East 49th St., Brooklyn 3, New York.

Boston University School of Medicine

The Oral Surgery Section offers a one-year oral surgery course planned to meet the requirements of the American Board of Oral Surgery. It provides a foundation for clinical training, such as clinical internship and residency, leading to qualification for the practice of oral surgery as a specialty. This course, given annually, is designed to give, by lecture, clinical demonstration, operative clinic, and laboratory exercise, a systematic review of the clinical phases of oral surgery. This is coordinated and correlated with the pertinent preclinical sciences. The fundamental principles of diagnosis and treatment of the various manifestations in the field of oral surgery are covered, with specific emphasis on etiology and pathology. Instruction is given in oral surgery, principles of general surgery, radiology, general and oral pathology, oral medicine, physical diagnosis, medicine, dermatology, clinical laboratory procedures, anesthesia, physiology, biochemistry, pharmacology, anatomy (head and neck), bacteriology, surgical prosthesis, and oncology. The course is under the direction of Dr. Kurt H. Thoma, Professor of Oral Surgery.

The Orthodontics Section offers a course of two academic years intended for those who wish to prepare themselves for the practice of orthodontics as a specialty. This course consists of lectures, clinical demonstrations, clinics, and laboratory exercises covering the various

phases of the specialty. Courses are given in the preclinical sciences pertaining to this field. Applications are being accepted for the program which begins in the fall of 1962. The course is under the direction of Dr. Herbert I. Margolis, Professor of Orthodontics.

The Prosthetics Section offers a one- or two-year course in general prosthetic dentistry. It is intended for those who wish to prepare themselves for the practice of prosthodontics as a specialty and/or who may wish to prepare for the specialty board examinations. This course consists of lectures, clinical demonstrations, operative clinics, and laboratory exercises in all clinical phases of the field. These are coordinated and correlated with pertinent preclinical sciences. The course is under the direction of Dr. Leo Talkov, Assistant Professor of Prosthetic Dentistry.

The Endodontics Section, offers a one-year course to dentists wishing to specialize in the field of endodontics. This course consists of lectures, clinical demonstrations, clinics, and laboratory exercises covering the various phases of this specialty. Instruction is given in the preclinical sciences pertaining to this field. The course is under the direction of Dr. Herbert Schilder, Assistant Professor of Endodontics.

The Oral Pathology Section offers a two-year course intended to prepare dentists for teaching or research in this field. The program includes the pertinent basic sciences and clinical oral medicine. The related curriculum consists of anatomy, biochemistry, physiology, microbiology, oncology, dermatology, general pathology, and oral pathology. The general pathology is given in connection with the Pathology Department of the Massachusetts Memorial Hospitals.

For application or information, please apply to Department of Stomatology, Boston University School of Medicine, 80 East Concord St., Boston 18, Massachusetts.

Albert Einstein College of Medicine
Yeshiva University

The Department of Dentistry will sponsor postgraduate courses in anesthesiology, endodontia, oral pathology, oral surgery, periodontia, and prosthetics during the spring of 1961. These courses are designed to be integrated into the general dental practice. Lecture sessions will be held at the Albert Einstein College of Medicine, and the clinical demonstrations will be given at the Dental Clinic, Jacobi Hospital.

For further information and application, please write to Director, Postgraduate Dental Division, Albert Einstein College of Medicine, Eastchester Rd. and Morris Park Ave., Bronx 61, New York.

University of Illinois

The College of Dentistry and the Graduate College of the University of Illinois have announced a graduate program in the field of oral histology and biology for qualified postdoctoral trainees who wish to prepare themselves for a career in dental teaching and research.

Further information can be obtained by writing to the dean of the College of Dentistry, University of Illinois, 808 S. Wood St., Chicago 12, Illinois.

University of Pennsylvania

The School of Dentistry announces the following postgraduate courses:

Applied Anatomy. Feb. 22 to 24, 1961. Harry Sicher.

Diagnosis and Treatment Planning. March 6 to 10, 1961. Lester W. Burket, Alvin L. Morris, and Roy T. Durocher.

Practical and Simplified Procedures in Crown and Bridge Prosthesis. March 13 to 17, 1961. Charles B. Scein.

Complete Denture Prosthesis. March 27 to 30, 1961. Vincent R. Trapozzano.

Endodontics. April 10 to 21, 1961. Louis I. Grossman and associates.

Minor Tooth Movement and Temporary Stabilization. April 12 to 14, 1961. Leonard Hirschfeld.

The twelfth Mid-Atlantic States Conference on Dentistry will be held at Buck Hill Falls, Pennsylvania, April 16 to 21, 1961. The theme of this year's conference is "Current Clinical Practices," and essayists will be H. J. Bartelstone, D. Walter Cohen, Orville B. Coolidge, S. Howard Payne, Samuel Seltzer, and Robert B. Shira.

For further information, please write Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

Oral Pathology Fellowship at University of Pennsylvania

Applications are being accepted for a clinical fellowship in oral pathology, supported by the American Cancer Society and tenable at the Department of Pathology, School of Dentistry, University of Pennsylvania, for a period of one year beginning July 1, 1961. This fellowship is open to United States citizens who are graduates of a United States dental school. A stipend of $3,600.00 (tax free) accompanies the fellowship. The program is planned to suit the specific needs and interests of the Fellow. Each candidate should submit a curriculum vitae and letters of reference. Interested persons are asked to contact Dr. C. E. Wilde, Jr., School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pennsylvania.

International Association for Dental Research

The International Association for Dental Research will hold its thirty-ninth annual meeting at the Statler Hotel in Boston, Massachusetts, March 23 to 26, 1961.

Approximately 300 papers will be delivered, and several symposia are planned for this meeting. The program will include such topics as dental caries (both human and animal), properties of enamel and dentin, wound healing, microbiology, nutrition and metabolism, and salivary studies.

Close to 1,000 persons, including about 400 IADR members, are expected.

The Dental Materials Group, composed of IADR members whose chief research interest is improvement of materials used in dentistry, will meet concurrently. About fifty research papers will be presented.

Western Society of Periodontology

The Western Society of Periodontology announces that its eighth annual scientific meeting will be held at the Tropicana Hotel in Las Vegas, Nevada, March 19 to 21, 1961. The guest speaker will be Dr. Robert F. Moyers of the University of Michigan.

For further information, write Dr. Daniel Feder, 152 South Lasky Dr., Beverly Hills, California.

University of Alabama

The School of Dentistry announces that a course in complete denture prosthesis will be given on March 10, 11, and 12, 1961, by Dr. William L. McCracken.

University of Texas Dental Branch

A course in pedodontics will be offered March 20 to 22, 1961, under the direction of Dr. Richard E. Jennings, Professor and Chairman, Department of Pedodontics. Dr. Ralph E. McDonald, Professor and Chairman, Department of Pedodontics, Indiana University School of Dentistry, will be the visiting lecturer, and Dr. Irvin A. Kraft, Chief of Psychiatry at Texas Children's Hospital, will be a guest speaker. This course is acceptable for 24 hours of credit by the Texas Academy of General Dentistry. Tuition for the course will be $75.00.

Further information may be obtained by writing to the Postgraduate School of Dentistry, The University of Texas Dental Branch, 6516 John Freeman Ave., Houston 25, Texas.

REVIEWS OF THE LITERATURE

BOOKS RECEIVED

Practical Orthodontics. George M. Anderson, D.D.S., Sc.D., formerly Professor of Orthodontics, Baltimore College of Dental Surgery, Dental School, University of Maryland, Baltimore, Maryland; Paul A. Deems, D.D.S., former Associate Professor of Clinical Oral Pathology, former Associate Professor of Dental Anatomy, and former Instructor in Clinical Orthodontics, Baltimore College of Dental Surgery, Dental School, University of Maryland, Baltimore, Maryland (collaborator); and contributors B. Holly Broadbent, D.D.S., D.D.O. (Glasgow), Sc.D. (Dublin), Director of the Bolton Study of the Development of the Face of the Growing Child, Laboratory of Anatomy, Western Reserve University School of Medicine, Cleveland, Ohio; Edward A. Kitlowski, M.D., Clinical Professor of Plastic Surgery, University of Maryland School of Medicine, Baltimore, Maryland, Chief of Plastic Surgery Service, Children's Hospital, Baltimore, Maryland; Bernhard Wolf Weinberger, D.D.S., Consultant Librarian, New York Academy of Medicine, Librarian, First District Dental Society, New York, New York, former Professor of Dental History and Literature, New York University College of Dentistry, New York, New York, Lecturer to Graduate Students, University of Pennsylvania School of Dentistry, Philadelphia, Pennsylvania; and Chester F. Wright, D.D.S., South Bend, Indiana. Ninth edition. St. Louis, 1960, The C. V. Mosby Company. 738 pages, 719 illustrations. Price, $18.00.

ABSTRACTS OF CURRENT LITERATURE

Cartilage Block Arthroplasty for the Correction of Temporomandibular Joint Disturbances. E. C. Hinds and A. H. Sills. Am. J. Surg. **98:** 787, December, 1959.

Cartilage block arthroplasty was found most suitable in cases in which a deformity, such as open-bite, unilateral shift, or retrusion, is associated with loss of the condyle.

Neurilemmoma of Bone. T. G. Sampter, F. Vellios, and W. G. Shafer. Radiology **75:** 215, August, 1960.

The authors describe neurilemmoma as a benign encapsulated tumor arising from the sheath of Schwann. It may arise centrally or in a nutrient canal of bone, occurring along the course of the peripheral, cranial, and sympathetic nerves.

The case of a 20-year-old Negro woman who had a swelling of the left side of mandible is presented. X-ray examination revealed a large region of destruction involving the ramus, the angle, and the posterior portion of the body of the left mandible. The cortex was expanded and thin. An excision biopsy was done, and the pathologist reported: "The predominant pattern was the Antoni type A. Verocay bodies were numerous. Antoni

type B tissue was present in small amounts. The patient was first seen in November, 1954, in 1955, and 1957. In July, 1960, the ramus and a portion of the body of the mandible was resected. Persistent neoplasm was found throughout the specimen."

Sialography and Surface Anatomy of the Parotid Duct. H. Oppenheim and M. Wirg. A. M. A. Arch. Otolaryng. **71**: 80, January, 1960.

There are many variations in shape and extent of the parotid gland, not only in different persons but also on the two sides of one person.

Sialography is of great value as an aid in the diagnosis of diseases of the salivary glands and associated structures. The technique is to inject 0.5 to 1 c.c. of Lipiodol or Iodochlorol into the unanesthetized orifices of the parotid duct through a blunt 18 or 19 gauge needle. Posteroanterior and lateral x-ray films are then taken.

An interesting description of variations in the duct and gland is presented in a series of 100 consecutive sialograms.

The Problem of Malignancy in Certain Neoplasms Often Considered Benign. A. C. Broders. Am. J. Digest. Dis. **54**: 384, April, 1960.

The author states: "I am of the opinion that mixed tumors under discussion, regardless of their duration, rate of growth, size or situation, are inherently malignant and should not be called benign."

One case is presented in which a tumor of the parotid had been present for fifty-three years and, on removal, was found to be infiltrated with carcinoma. Several cases are reported which should prove interesting reading to the oral surgeon.

Radiation Therapy of Carcinoma of the Tongue. E. M. Uhlmann and M. E. Weiner. Surg. Gynec. & Obst. **111**: 363, September, 1960.

Carcinoma of the tongue is the most common of all malignant lesions of the oral cavity. The disease occurs more frequently in men than in women. The treatment of cancer of the tongue originally was limited to surgery. The development of radiation therapy during the last three decades and its application in the treatment of cancer of the tongue resulted in the improvement of end results, with avoidance of the mutilation associated with surgery.

This report is an attempt to evaluate the results of radiation therapy in the management of cancer of the tongue and to establish a base for comparison with results achieved by surgery alone. It is recommended reading for the oral surgeon who has the opportunity to refer patients with proven cancer for treatment.

T. J. C.